THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. XV. OCTOBER, 1934 No. 58

97

Original Papers

OBSERVATIONS ON RECORDS OF LOCAL EPILEPTIC CONVULSIONS *

By

D. DENNY-BROWN AND E. GRAEME ROBERTSON, LONDON

PAGE

INTRODUCTION 97

METHOD 98

PROTOCOLS 98

DISCUSSION 119

The Cerebrospinal Fluid Pressure 120

The Effect of Sleep 121

'Reflex' Epilepsy 122

The Incoordination of the Epileptic Discharge 124

The Periodicity of Seizures 131

CONCLUSIONS 135

INTRODUCTION

THE varied phenomena of epilepsy and the many clinical problems with which it is associated present a perennial source of interest to the clinician. The convulsion itself presents many problems which become the more interesting the more closely they are observed, though the rapid sequence and concurrence of a number of manifestations tend to confuse clinical observation. Some of the events lend themselves to analysis by graphic record, and the following account concerns records of cerebrospinal fluid pressure, respiration, and muscular movement in six patients. Though such records fall far short of any exact quantitative measurement of muscular contraction, careful control by clinical observation allows them a measure of accuracy, and in their essential feature, namely sequence of events in time, they appear to us to be reliable evidence.

* From the National Hospital for Nervous Diseases, Queen Square, London.

XUM

METHOD

The pressure of the cerebrospinal fluid was recorded by means of a mirror tambour capsule which reflected a beam of light to a moving strip of bromide paper. The pressure was transmitted from a wide lumbar puncture needle to the glass capsule by means of thick rubber tubing of small inside diameter. A small sterile chamber intervened between the needle and the tubing, and here the cerebrospinal fluid reached a level by compressing the air within the capsule system. The capsules used are identical with those we [5] have described in detail for use in the measurement of vesical pressure, and are calibrated after each experiment by measuring the deflection produced by water columns of various heights. The periodicity of the system is well damped by the slight fluid movement ; the latency is negligible at the rates of recording used. The patient lay on the least active side, on a wide couch, for periods up to three hours. Movements have been recorded by the attachment of pressure balloons over the chest wall, and over the muscle concerned, with due precaution to avoid the possibility of movement by other muscles. Changes in pressure within the balloons was transferred to capsules with mirrors which recorded on the same bromide paper. The record of movement, though sufficiently accurate for time and general amplitude, cannot be used for accurate quantitative analysis. The latency of movement was tested separately and found to vary from 0·004 sec. with short tubing (head, trunk and upper limb) to 0·01 sec. with longer tubing (lower limb). In records as rapid as those in figs. 25–29 there was no measurable difference between air transmission and direct beam from muscle. Time was recorded by a rotary Rayleigh wheel driven by an electrically operated tuning fork. When action currents have been recorded a Matthews oscillograph system has been used, with leads from the muscle by needle electrodes.

PROTOCOLS

Case I. Regular series of predominantly left-sided convulsions at brief intervals. Previous history of sensory and inhibitory attacks. No loss of consciousness. Pain in affected limbs. Spastic left-sided weakness due to right parietal astrocytoma.

M.B., age 54 years, female, was admitted under the care of Dr. Gordon Holmes in November, 1932. There was no pertinent family history or previous illness. In September, 1922, there occurred the first of a long series of brief attacks at intervals from several days to several weeks. In each of these there occurred a sensation of something rushing over her head, lasting several seconds, during which she could neither speak nor move. There was no loss of consciousness, and she would be observed only to stand still with fixed expression. At times the patient would turn round during an attack, and on some occasions the left arm and leg would become somewhat stiff, at times only the left leg. In other attacks there was a pricking sensation of the left side of the neck and tongue. The frequency of these attacks was much reduced by luminal, though they continued at long intervals for the next three years.

In one night in May, 1925, there occurred four generalized convulsions with loss of consciousness and preceded for a short interval by a severe pain in the left leg ' as

though someone were trying to pull it out of its socket.' She was not incontinent nor did she bite her tongue. She was admitted to another hospital and a right parietal decompression performed, after which the left arm and leg were completely paralysed but recovered in four weeks. Attacks of varied pattern continued. For a brief interval she had warnings of the 'wave passing over her head,' but from that time onwards she had no aura. There were attacks of tonic and then clonic spasm in the left arm and leg, attacks in which she was unable to use the left arm for a few seconds, and also attacks in which the left leg would feel quite numb for perhaps a half-minute. In none was there loss of consciousness, and the attacks recurred at intervals of a few hours to a few weeks, the intervals being longest when she was taking luminal.

On November 15, 1932, there developed a numbness of the left arm and leg, which lasted continuously for five days, and the patient then commenced to have very frequent attacks of spasm of flexion, lasting 15 seconds to one minute, at the left hip and knee, accompanied by severe pain in the left thigh. As the attacks continued progressive loss of voluntary power in the left lower limb occurred. There was no involvement of the right side. At the onset of later attacks the left arm was drawn up, but she was still able to move the fingers.

Attacks observed on November 22, 1932, presented the following features: If the patient were talking speech ceased suddenly and her face stiffened. There was a variable degree of stiffening of both arms and the right leg; the left lower limb became tonically flexed at the hip and knee with fine jerking movements at the ankle. After some 10 to 12 seconds the jerking increased in amplitude and clonic movements at the hip and knee commenced and lasted about 15 seconds. In some attacks the left arm adducted strongly. The patient was able to move the right arm to order during the attacks and stated that she was fully conscious and suffering pain in the dorsum of the left foot and in the left thigh.

There was slight bilateral papillœdema and slight weakness of voluntary movement of the left side of the face. All movements in the left upper limb, especially those at shoulder and elbow, were weak, and there was spasticity in the adductors of the shoulder and flexors of wrist and elbow. There was no power of voluntary movement in the left lower limb, the extensors of which were extremely spastic. The tendon reflexes of the left limbs were exaggerated and the left plantar response was extensor. There was no defect of sensation. The decompression was under no increase in tension. The cerebrospinal fluid showed no abnormality.

She was treated by luminal gr. 2 three times daily. On November 20 she had 60 attacks, the following day 30, and on the day of examination 50 attacks. Her temperature was irregular, rising from 97·4° to 99·6° F.

Subsequently, on November 25, the previous decompression opening was explored by the late Sir Percy Sargent and the cerebral cortex of the parietal lobe was found to be replaced by gelatinous tissue which extended to the median longitudinal fissure. A mass about 2 inches in diameter was removed and histological examination proved it to be an astrocytoma. She made a good recovery from operation and was discharged from hospital with a spastic weakness of the left arm and leg.

Analysis of attacks recorded on November 22, 1932.—In this patient besides a manometer to record the pressure of the cerebrospinal fluid through the lumbar puncture needle it was arranged that the movements of the upper abdomen and of the left great toe were recorded, together with the fluctuations in pressure at the decompression opening on the right side of the head. We had the kind assistance of Dr. Gilbert Hall in recording the attacks. Seven fits were recorded in their entirety, and the characteristic records shown

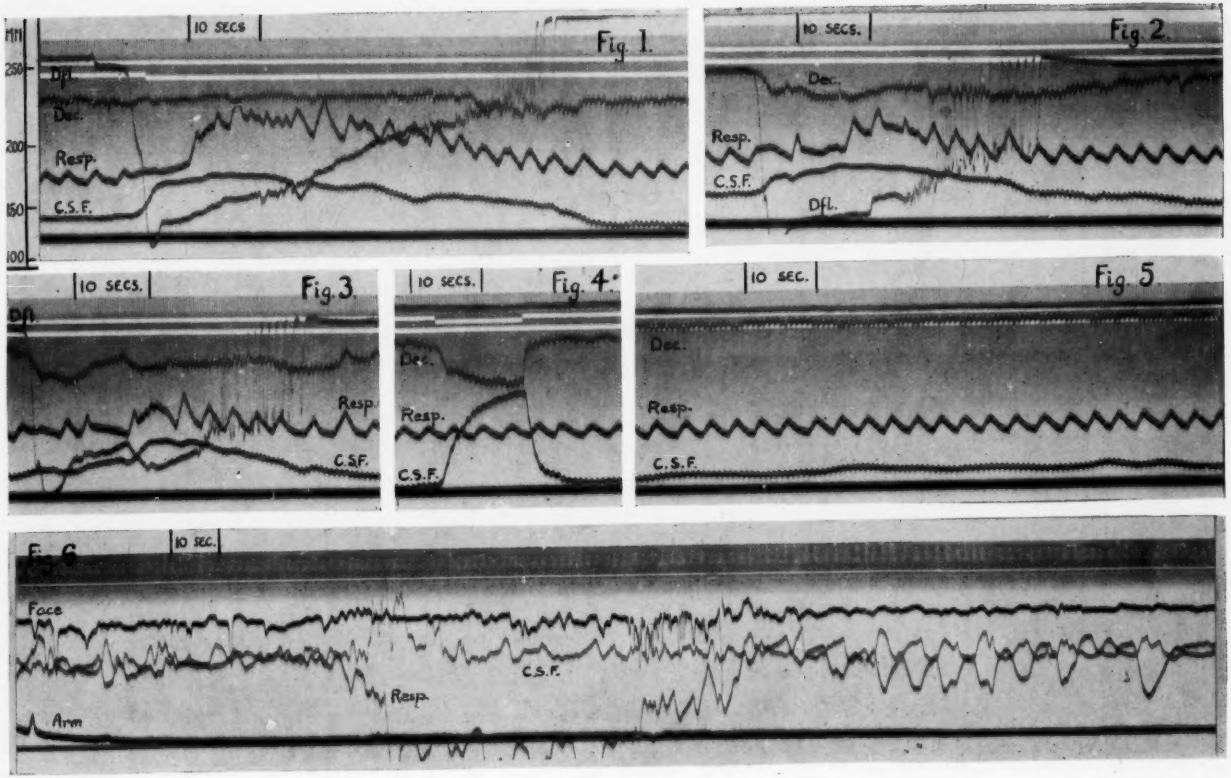

mm
250
200
150
100
10 secs
Fig. 1.
Dpl.
Dec.
Resp.
C.S.F.
10 secs.
Fig. 2.
Dec.
Resp.
C.S.F.
Dpl.
10 secs.
Fig. 3.
Dpl.
Resp.
C.S.F.
10 secs.
Fig. 4.
Dec.
Resp.
C.S.F.
10 sec.
Fig. 5
Dec.
Resp.
C.S.F.
Fig. 6
10 sec.
Face
C.S.F.
Resp.
Arm

PLATE I.

in figs. 1, 2 and 3 show the course of the attacks. As will be seen in all these figures the onset of the attack was an abrupt dorsiflexion of the great toe, which was in all instances succeeded by a momentary cessation of respiration shown by the abdominal recorder. The cerebrospinal fluid pressure commenced to rise, usually as early as four-fifths of a second after the onset of dorsiflexion of the great toe (figs. 1 and 2), although in isolated instances, such

---

DESCRIPTION OF PLATE I

Plates I–VII. From records of epileptic attacks made photographically by movements of beams of light on a moving strip of bromide paper. All records read from left to right. Time is recorded electrically in fine vertical strokes at the top of the record at one-fifth second intervals. As the closeness of the vertical strokes in slow records tends to make them illegible after reduction, intervals of 10 seconds have been superimposed for the convenience of the reader. In Plates VI and VII the speed of the record renders this unnecessary. The tracing of the manometer recording cerebrospinal fluid pressure is marked C.S.F. in all records. In figs. 8, 11, 13, 14 and 15 the cerebrospinal fluid pressure is recorded both by delicate and coarse manometers the tracings of which are marked C.S.F. 1 and C.S.F. 2 respectively. The scale of tension at the commencement of each record refers to the calibration of the cerebrospinal fluid pressure (in mm. of water) as registered by the delicate manometer. The tracing which records chest movement is marked Resp. (or in some cases where abdominal movement was recorded, Abd.). The deflection is downwards for inspiration in all cases. In each case a record is made of some muscular movement prominent in the attack and the lettering of these records is described in the legend of each figure.

Plate I. Fig. 1.—Record of epileptic attack in Case I, 12 minutes after the last preceding attack. The uppermost moving line Dfl. registers movements of the left great toe and makes downward excursion for dorsiflexion and small upward excursion for plantar flexion. The tracing Dec. records the pressure within a balloon bound over the decompression opening in the skull, and shows an excursion with each pulse beat, and was also affected by movements of the face and neck. The pressure within the balloon at the commencement of the record was 12 cm. of water. The small movement of the foot three seconds before the commencement of a fit was probably voluntary.

Fig. 2.—Tracings and C.S.F. tension scale as in fig. 1, here showing a fit of shorter duration, 10 minutes after that recorded in fig. 1 and two and a half minutes after an intervening fit. The higher initial cerebrospinal fluid pressure was consequent upon some apnœa occurring after the hyperventilation of the intervening fit.

Fig. 3.—Tracing and C.S.F. tension scale as in fig. 1. Two minutes 40 seconds after the attack featured in fig. 2, showing delayed rise in cerebrospinal fluid pressure.

Fig. 4.—Tracings and C.S.F. tension scale as in fig. 1. To show the effect of compression of the left jugular vein in the interval signalled by the duration of the fall in the upper white line.

Fig. 5.—Tracings and C.S.F. tension scale as in fig. 1. To show irregular fluctuations in the cerebrospinal fluid pressure during a period of drowsiness some 10 minutes after the attack recorded in fig. 3.

Fig. 6.—Showing myoclonic twitching followed by major attack in Case II. The uppermost darker tracing (Face) shows a downward deflection for contraction of the left corner of the mouth. The figure commences with the occurrence of the first movement after a long period of quietude. Following the period shown in this figure the twitching disappeared after a few seconds and hyperpnœa occurred before the patient again became quiet. In the centre of the figure contraction of the trunk muscles in the tonic stage of the convulsion causes prolonged displacement of the chest recorder (Resp.). C.S.F. pressure as in scale in fig. 7.

PLATE I.

XUM

as fig. 3, the rise was much longer delayed. This rise of pressure amounted at times to as much as 65 mm. of cerebrospinal fluid, as in fig. 1. After a first rapid rise it usually maintained an almost steady plateau, commencing to subside only when abrupt irregularity in the toe-recorder showed the first evidence of the clonic stage. Whether regular beats of clonus continued from this time onwards, as in fig. 2, or became established only very late in the attack, as in fig. 1, the reestablishment of original cerebrospinal fluid pressure was gradual and progressive. As will be seen in all three records the later fall of fluid pressure is related more directly to the reestablishment of regular respiration, as shown by the abdominal recorder. Where the abdominal movement at the onset of the attack is slight, as in fig. 3, the cerebrospinal fluid pressure may rise rapidly, late in the tonic stage, when the abdominal and thoracic muscles eventually participate in the attack.

The attempted record of the pressure at the decompression opening was found to be partly independent of the cerebrospinal fluid pressure change, as is seen in the figures shown, and inspection at the time revealed that, however applied, the fixation of the balloon at the decompression orifice was affected by the powerful movement of the face and neck musculature, which in most attacks commenced to occur within from three-fifths to one second after the first movement of the great toe. The absence of any real independence in cerebrospinal fluid and intracranial pressure is shown by light jugular compression when particular care was exercised not to disturb the fixation of the balloon over the decompression (fig. 4).

It will be noted that the resting cerebrospinal fluid pressure varied from 135 to 150 mm. of fluid owing to the continual fluctuations which were observed during the intervals between fits. These were particularly marked when the patient was drowsy, as in fig. 5, but in this patient were not observed to be greater than 20 mm. in range. They were not accompanied by any parallel change in respiration or abdominal posture. In one attack the pressure following the fit remained higher (110 mm.) than it was before the attack (100 mm.). In the other attacks, as in figs. 1, 2 and 3, the pressure was lower for an interval of some 15 to 30 seconds following the attack. There was no change preceding the attack in any of the records.

Case II. Generalized convulsions from early age. Three periods of status epilepticus at long intervals. Epileptic myoclonus, with 'reflex' provocation. Major attacks with predominantly left-sided convulsion. Repeated development and recovery of postepileptic hemiplegia.

G.T., age 12 years, female, admitted under the care of Dr. Gordon Holmes, in July, 1931, May, 1932, and January, 1933. No family history of epilepsy. Generalized convulsions commenced at the age of four years, and continued at the rate of two to four a week. The attacks were almost always nocturnal, usually with biting of the tongue and rarely with incontinence. Luminal administered regularly controlled the attacks, but the parents refused to continue its use. In July, 1931, generalized

convulsions, with loss of consciousness, incontinence and biting of the tongue, commenced to occur at hourly intervals. There was sufficient recovery in the intervals to talk, but after two weeks the left upper and lower limbs were noticed to be weak between the attacks. A week later, when she was admitted to hospital, the attacks were still occurring at the rate of about one an hour, each followed by a drowsy stage which lightened only for a brief interval before the onset of the next attack. The left limbs were weak, particularly in the grasp of the hand and in dorsiflexion of the foot, and both were slightly hypertonic, with increased tendon jerks. The abdominal reflexes were absent on the left side and the left plantar response was extensor.

In the attacks the head was turned to the left and the eyes upwards and to the right, the left arm and leg showed clonic movements, the right arm and leg rigidity. In some attacks twitching of the face preceded that of the left arm and leg. The evening temperature was usually elevated—on the thirteenth day, when the fits were lessening, reaching 102° F. Only once was the morning temperature high (99° F.). There was no correspondence between the number of attacks and the height of the temperature. She was treated by luminal and paraldehyde (fig. 30, status I). The intervals between attacks lengthened and the left hemiparesis gradually disappeared.

On discharge from hospital the patient remained free from attacks for one month and at the end of that period, at intervals of seven to 14 days, there commenced a series of momentary nocturnal attacks of twitching of the eyelids with generalized stiffness and trembling. Nocturnal enuresis was frequent. At the beginning of May, 1932, the frequency increased to two to three attacks every night and within a few days attacks became very frequent indeed, and generalized convulsions occurred. The smaller attacks now occurred in the daytime. In these, with the trembling there was a little cry, the lips would become blue, and she was unable to talk for a moment or two. The large attacks took the form of turning of the head to the right with flexion of the arms at the elbows, hands clenched, eyes closed, with some cyanosis of the lips. The head then turned to the left and the left arm extended. The attack subsided with some clonic jerking of the left arm and was followed by drowsiness. The total duration was some 30 seconds. In other attacks the head turned to the left with eyes to the right, the right upper limb was held extended, the left flexed. In the intervals between the attacks the left arm and leg were hypotonic, but there was at this time no defect in motor power, except for a left facial weakness. The tendon reflexes were increased in the left upper limb, but equal in the lower limbs. Abdominal reflexes were absent and plantar responses both flexor. Blood pressure 120/70.

Neither paraldehyde nor morphia controlled the attacks, which were temporarily reduced in number, but not controlled by luminal gr. 5 intravenously. The attacks diminished in severity but the total frequency was little altered. A left-sided hemiparesis developed, with marked left-sided hypotonia, and extensor plantar response. There was defective sensation of position and passive movement with astereognosis in the left hand. At this time records of the fits were made. The temperature rose only on two occasions, once in the evening to 99° and once in the morning to 99·8° F. (fourteenth and sixteenth days—fig. 30, status II). Treatment by bromides, chloral and luminal (up to gr. 4 a day by mouth) was continued and the number of attacks lessened and eventually ceased. Ventriculography showed no displacement or deformity of the ventricles.

Attacks continued at very rare intervals during the next six months, when again, in spite of continuous treatment by luminal, tonic convulsions of the left arm and leg commenced to become very frequent. On this occasion a left-sided hemiparesis, with flaccid arm and slight increase of tone in the lower limb, developed. There was loss of two-point discrimination and astereognosis in the left upper limb. The attacks were as before, except that both head and eyes were turned to the left. Respiration was profoundly affected. The left plantar response was observed on several occasions

XUM

to be extensor for a period of three minutes after an attack, the right remaining flexor throughout. The attacks gradually lessened in frequency and finally ceased (fig. 30, status III). The temperature showed only occasional elevation to 99·2° F. on the morning of the third day and to 99° F. on the evening of the twenty-sixth day. When the attacks were most numerous the temperature varied between 97° and 98·4° F.

On March 7, 1933, an exploration of the cerebral cortex by a right fronto-temporo-parietal osteoplastic flap was made by Mr. Julian Taylor. The subarachnoid lakes appeared to be unusually distended. There was no evidence of tumour. The patient made a rapid recovery, and has remained free from fits.

Analysis of attacks recorded on June 10 and 15, 1932.—The facial movements in this patient showed continued irregular brief twitchings occurring singly or in groups. At times twitching of the left arm and of the left side of the trunk also occurred. In records of these twitchings there appeared to be no direct relationship between the occasional myoclonic twitches in the face and those which were observed in the trunk and wrist as shown in figs. 6 and 8. At long intervals, usually when all twitching had gradually subsided in the preceding 10–20 minutes, abrupt and violent twitching would break out in the left side of the face, trunk and left arm concurrently. This would partly subside sometimes after five or 10 seconds, leaving for an interval more frequent twitching in the face. The cerebrospinal fluid pressure at these times showed only some small fluctuations corresponding to those of the chest. At other times, as in fig. 6, the initial abrupt movements in the face and trunk gave way to a more maintained tonic posture lasting up to a minute, during which time some isolated jerky movements of the face, arm and trunk occurred. For a variable period following this a rapid irregular clonic twitching of the face remained. With the initial outburst in such cases the cerebrospinal fluid pressure showed irregular jerks of increase, but fluctuations occurred corresponding in time to the larger trunk movements. In the intervening periods when the patient was drowsy, when myoclonic twitching was rarely observed or at times absent for periods of minutes, it was noticed that a sudden stimulus caused awakening accompanied by a rapid sequence of myoclonic twitches. In this way a myoclonic attack or even a tonic-clonic convulsion appeared to be elicited by stimulation. Such a convulsion is recorded in fig. 7. Here, a loud noise having proved ineffective, sudden cold was applied to the left calf. Immediately there resulted movement of the left arm and left leg and after two seconds rapid twitches of wide amplitude appeared in the left side of the face and the left limbs. These ceased after a further two seconds, when there was a powerful contraction of the left side of the trunk with cessation of respiration. After a cessation for some four seconds this tonic contraction was renewed in more powerful form and a tonic convulsion was established. The clonic stage which ensued passed into irregular myoclonic twitching lasting many minutes. The cerebrospinal fluid pressure undergoes small fluctuations which reflect only the more abrupt movements of the chest.

XUM

Case III. Two isolated outbursts of status epilepticus, with long intervening interval. Compulsion aura. Predominantly right-sided attacks with speech defect. Postepileptic paralysis. Asthmatic subject.

L. K., male, age 31 years, admitted to the National Hospital October, 1933, under the care of Dr. Hinds Howell. From his youth he had suffered from attacks of asthma, with occasional eruptions of giant urticaria. In October, 1932, without warning he had a generalized convulsion, with loss of consciousness, succeeded by stupor, which was interrupted 24 hours later by another convulsion. During the next nine days, of which he has no memory, he remained in a stuporose state. He states that in the subsequent few days he felt 'completely exhausted' and remembers having six to eight attacks in which his right arm jumped and the elbow flexed involuntarily. He then remained free of attacks until April, 1933, when he commenced to experience momentary 'sensations' in which he felt that he 'must do something but did not know what.' Two weeks later one of these sensations was followed by a generalized convulsion with loss of consciousness. Treatment with luminal was then commenced and he had no further attacks until a generalized convulsion followed two weeks after the discontinuing of such medication in June, 1933. He was admitted to hospital five days later and no abnormal physical signs were found. The cerebrospinal fluid was normal. Skin tests showed a moderate reaction to the meat group, in particular to beef. On discharge he remained well except for periodical asthmatic attacks, which were relieved by ephedrin. At the height of one such attack the occurrence of one of his 'sensations' was accompanied by subsidence of the asthmatic attack. On September 26, 1933, after momentary confusion of mind he lost consciousness for a period of two hours, after which he remembers twitching all over. His asthmatic trouble, previously occurring nightly, was lessened for 48 hours. Four days later the patient had an attack in which he felt impelled to say something but was unable to speak, following which his right arm was raised. Similar attacks followed, and in the intervals, which became progressively shorter, he felt exhausted and had difficulty in expressing himself. In this state, two days later, on October 2, when the attacks were recurring every 20 minutes, he was admitted to hospital. The unpleasant sensation of necessity for speech, with the production of an unrecognisable sound which trailed off into 'a silence,' continued to precede each attack. He was able to understand what was said to him during an attack but could make no response of any kind. Observed attacks commenced with an irregular groaning, twitching of the upper lip, turning of the head to the right, and similar deviation of the eyes with occasional reassertions of forward gaze. His right arm became flexed at elbow and wrist. After two minutes the periodic groaning ceased, he would smile, and after a momentary clonic twitching of both corners of his mouth, he would turn his head forward and speak rationally. A series of such attacks was recorded.

Upon examination slight weakness of the lower right side of the face and of the right side of the palate and tongue were apparent. Motor power, tone and limb reflexes were normal, except that the right plantar response was less flexor than the left, and immediately after an attack was extensor for a few minutes. The cerebrospinal fluid contained 20 cells per c.mm., total protein 0·030 per cent.; globulin tests were negative and the Lange normal. Ventricular fluid contained 19 cells per c.mm. and protein 0·03 per cent. Ventriculography failed to reveal any abnormality of the cerebral ventricles.

The day after his admission some attacks involved the face alone, and while the right side was drawn up in spasm the left eye would close intermittently and the mouth would be drawn first to one side and then to the other. In some attacks the right arm was involved in clonic convulsion, in others in tonic flexion with tonic extension of the

XUM

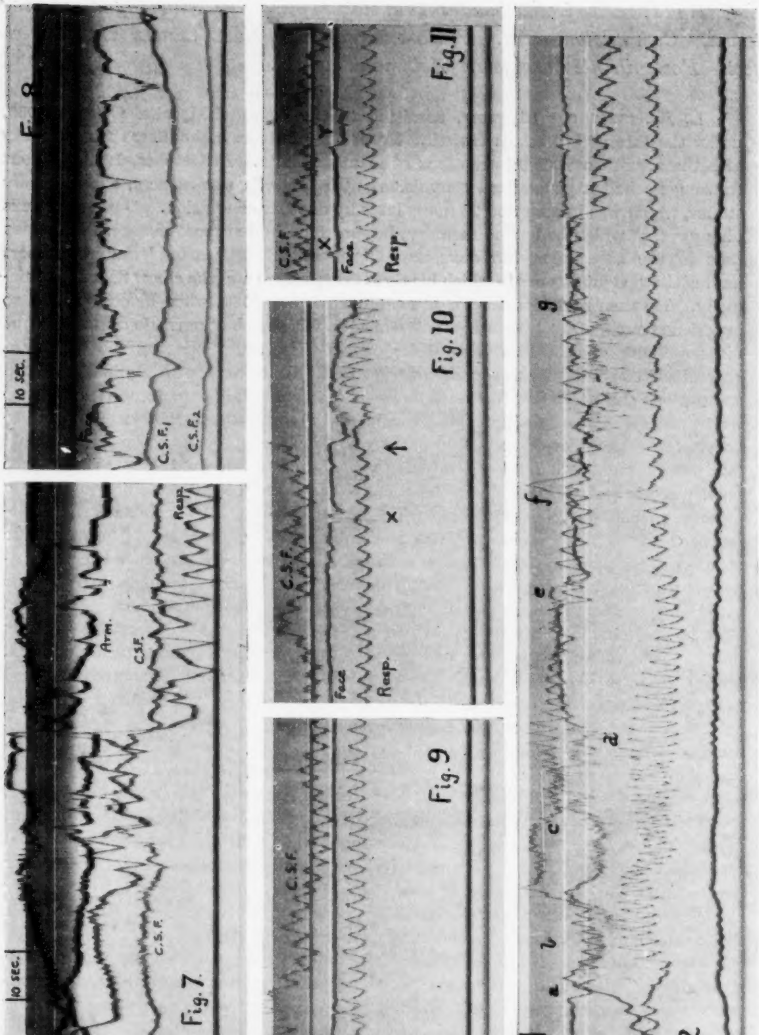

Face
Arm.
Resp.
C.S.F.
10 sec.
Fig. 7
C.S.F.
C.S.F.1
C.S.F.2
Fig. 8
Face
Resp.
10 sec
Fig. 9
Fig. 10
Fig. 11
Fig. 12
a
b
c
d
e
f
g
200
150
100
50
mm

PLATE II.

right leg. After some attacks he was completely aphasic for 10 minutes. Examination now showed increased weakness of the right side of the face, palate, tongue and right hand. The right arm was hypotonic and both lower limbs showed a slight increase in extensor tone. The tendon reflexes were equal and brisk in the lower limbs, with patellar clonus; the plantar responses were extensor during the attacks but plantar flexion replaced this reaction in the intervals. On the day of admission he had 33 attacks and three drams of paraldehyde were administered per rectum. The following day he had 26 attacks and was treated by two hypodermic injections of morphia (gr. $\frac{1}{6}$ and gr. $\frac{1}{4}$), 4 drams of paraldehyde per rectum, and luminal gr. $1\frac{1}{2}$ three times daily. The last was alone continued thereafter. On October 4 he had nine attacks, on October 5, five, and then was free from attacks during the rest of his stay in hospital. There was no elevation of temperature with the attacks.

Analysis of attacks recorded on October 2, 1933.—In this patient the use of a wide-bore lumbar puncture needle and a sensitive manometer enabled us to obtain a more than usually clear record of the pulse and respiratory waves. The close correspondence between the movements of the chest and the cerebrospinal fluid pressure is evident in fig. 9, where for a brief interval the patient is instructed to cough. Even speech, to the extent of the enunciation of one word, also causes a brief rise of pressure (fig. 11). The initial fall, and after a brief interval the sudden rise in cerebrospinal fluid pressure, are distinctly seen, also the subsequent slight fall of pressure for a period of some 30 seconds due to the increased movement of the chest with increased ventila-

PLATE II.

DESCRIPTION OF PLATE II

Plate II. Fig. 7 (Case II).—As in fig. 6, with the addition of a tracing (Arm) recording occurrence of flexion of the left wrist as an upward excursion. The patient having previously been asleep, cold is applied to the left calf at the moment indicated by the arrow directed upwards. A powerful myoclonic jerk is followed after a few seconds by the commencement of an attack in which tonic movement of the face with expiration is followed by a long period of myoclonus.

Fig. 8.—Tension scale for C.S.F. 1 as in fig. 7. The upper record shows by downward deflections a series of myoclonic contractions of the left side of the face. Respiration is not recorded.

Fig. 9 (Case III).—To show the effect of a cough in the duration of fall of the upper white line.

Fig. 10 (Case III).—Time, C.S.F. pressure, and respiration as in fig. 9. To show the earliest change in C.S.F. pressure as recorded by an extremely sensitive manometer. A balloon over the right corner of the mouth causes a manometer tracing (Face) to move downwards when contraction occurs. At the point marked X the patient made some small voluntary facial movement while at the point marked (↑) a convulsion commences.

Fig. 11.—C.S.F. tension, etc., as figs. 9 and 10. To show effect of two brief periods of speech marked X and Y.

Fig. 12.—Complete record of a fit in Case III. Time, C.S.F. tension and other records as in figs. 9, 10 and 11, with the addition of a tracing of a coarse manometer C.S.F. 2. Some momentary displacements of the upper white line are made by an electrical signal by the observer to indicate at *a* slight turning of the head towards the affected side, at *b* the first groaning sound, and at *c* more powerful turning of the head to the affected side; at *d* the patient nodded his head and said 'I'm all right.' From *e* to *g* the patient talked and at *f* coughed to order.

XUM

tion immediately following the apnœa. Jugular compression was followed by a rapid rise of cerebrospinal fluid pressure within two seconds. This manometer showed well the earliest changes during a fit (fig. 10), and a fully recorded fit with both coarse and delicate manometers is shown in fig. 12. The pressure is seen to commence to rise apparently as the first event in the attack, although close inspection of the record reveals that the accompanying respiration is more brief than its predecessors. The tonic stage of the attack lasts only five seconds in the face, and the steep rise of cerebrospinal fluid pressure during this interval thereafter ceases, falling very slightly during subsequent clonus and rising sharply again 15 seconds later where the facial movements again become tonic for a brief interval. The period of apnœa here outlasts the initial rise of pressure and at the end of the attack the pressure falls only very slowly, being delayed for some 20 seconds by a renewed outburst of clonic twitching of the face. The original level was reached only after four minutes from the termination of the attack. The recorded signals during this fit indicate that vocalisation occurred with or slightly after the first movement of the face and that the secondary rise of cerebrospinal fluid pressure begins seven seconds after the commencement of turning of the head. It will be noted that the regular rapid movements of the chest show no change at this time. All these features were confirmed in the record of another fit.

Case IV. Onset of epilepsy three months after the evacuation of a frontal abscess. Three periods of status epilepticus. Left-sided attacks, with post-epileptic paralysis and sensory disturbance. Coincidence with reflex stimulation.

L. S., age 27 years, female, admitted under the care of Dr. James Collier in March, 1930, January, 1931, and November, 1933. In 1922 the patient had a septic appendix removed. In 1925 there began severe frontal headaches with failing vision. In January, 1926, she was admitted to St. George's Hospital where a right frontal cerebral abscess was drained. In March, 1926, she commenced to suffer from attacks of unconsciousness preceded by twitching of the left arm and leg, occurring at long intervals for four years, but gradually increasing in frequency. Two days before her admission to hospital on March 14, 1930, attacks involving the left arm and sometimes the left side of the face commenced to occur every few minutes. For one to two minutes after the cessation of such attacks the left arm was useless. The attacks were partly controlled by the administration of four-hourly doses of paraldehyde and ceased five days after admission. She had only one rise of temperature, to 99·8° F., during this time. The attacks then ceased completely, but bromide and luminal were continued. In January, 1931, attacks of shaking of the left arm lasting a few seconds and occurring every half-hour commenced. Three days later the attacks spread further; commencing with flexion of the left elbow and wrist and turning of the head to the left, the left side of the face and trunk were affected later in the attack. After the onset of the fit there was no movement of the head, which could be turned in the opposite direction to order. There was no deviation of the eyes. Consciousness was not lost. Again weakness of the left side of the face and the left arm, with diminished tone and slight weakness of the left arm, was observed in the intervals between the

attacks. There was complete astereognosis, loss of two-point discrimination and diminution of postural sensibility in the left hand.

The attacks were treated by paraldehyde and rapidly became fewer, and thereafter under luminal treatment she remained free from attacks until October, 1933, having had a pregnancy in this interval. A single nocturnal fit then occurred. A week later, after a day of infrequent attacks, the fits recurred every three to five minutes. Paraldehyde treatment controlled the attacks to some extent until the fifth day after their onset, when they commenced to occur every two to five minutes. The attacks continued during waking and sleeping hours for 14 days in spite of intravenous administration of luminal gr. 3 on three occasions and 2 drams of paraldehyde by rectum four-hourly. During this period there gradually developed a complete flaccid paralysis of the left side of the face, arm and leg. In the three weeks following cessation of the attacks the paralysis slowly improved, leaving slight weakness of the left side of the face, astereognosis in the left hand and loss of two-point discrimination in the left hand and left foot. The temperature reached 99° F. on one occasion only, after the cessation of the attacks.

At the time records were made of the attacks the sequence of events was as follows: For a second or two the patient appeared to be aware that an attack was imminent. Her head turned slightly to the left and she complained of a tingling sensation in the left hand. The left arm was abducted at the shoulder and partially pronated at the elbow, with extended wrist. The wrist then flexed and the fingers extended. The left leg was then abducted at the thigh and feet were inverted and extended. The right leg showed a similar movement of the foot only. The left side of the face was slightly contracted. When these attitudes had been maintained for 10 to 15 seconds clonic movements appeared in the left side of the face, in the left arm, abdominal muscles and slightly in the lower limb. Clonic movements were sometimes seen in the deep muscles of the right side of the neck. These movements appeared to occur in the same order as the tonic spasm and persisted longest, sometimes in the left hand, sometimes at the left corner of the mouth. The patient was conscious and able to speak during the attacks.

In the intervals between the attacks the left arm and leg were in a state of complete flaccid paralysis. There was no demonstrable weakness of movements of the upper face, but the left side of the lower face remained completely immobile upon attempted voluntary movement. For associated movements the whole left side of the face moved well but not so strongly as the right. On emotional expression the left side of the lower face moved earlier and more completely than the right. The palate was slightly weak on the left side and the tongue deviated to the left. There was no weakness of sternomastoids or trapezil. There was slight weakness of the abdominal muscles on the left side. The tendon jerks were increased in both arms, and both knee and ankle jerks were present and equal. The abdominal reflexes were absent on the left and present on the right. The plantar reflex was flexor on the right, absent on the left. In the upper and lower limbs there was complete loss of position sense, appreciation of passive movement, stereognosis and two-point discrimination, with loss of position sense and two-point discrimination in the left lower limb. The blood pressure was 170/110 mm. Hg. both during and between the attacks. The cerebrospinal fluid was reported to contain no cells, protein 0·050 per cent., Nonne-Apelt negative, Pandy positive. Lange and Wassermann reactions negative. The blood calcium was 10 mgm. per cent. in one period of attacks.

Analysis of attacks recorded on November 7, 1933.—In this patient attacks were occurring at brief intervals of surprising regularity. In consecutive attacks which were recorded the intervals from onset to onset were as follows:

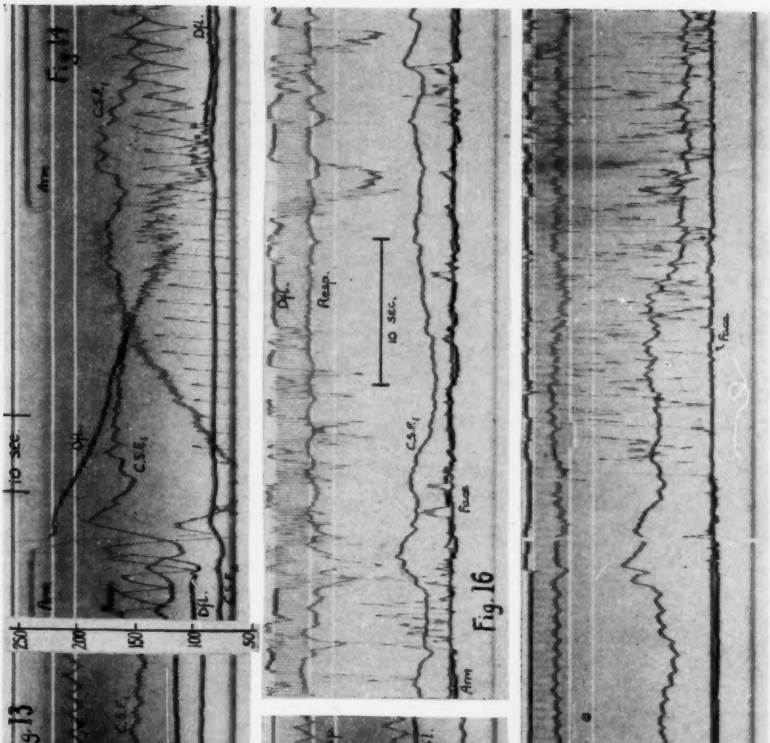


Fig. 13
Fig. 14
Fig. 15
Fig. 16
Fig. 17
10 sec.
Dyl.
Resp.
Arm
Fem
C.S.F.
C.S.F.1
C.S.F.2
a
b
c
d

PLATE III.

3 min., 3 min. 8 sec., 3 min. 8 sec., 3 min. 30 sec., 3 min. 54 sec., 3 min. 36 sec., 3 min. 36 sec.; after two attacks in which the interval was not recorded, a further consecutive series showed the following intervals: 4 min. 3 sec., 3 min. 22 sec., 3 min. 25 sec., 3 min. 37 sec., 3 min. 32 sec., 3 min. 28 sec., 3 min. 12 sec., 3 min. 28 sec., 3 min. 48 sec., 3 min. 32 sec. The slight variation was not accounted for by variation in the duration of each attack, which showed independent slight fluctuation.

In these attacks, examples of which are shown in figs. 13, 14 and 15, there was constantly an initial fall in cerebrospinal fluid pressure followed by a rapid and high rise, which thereafter subsided gradually during the clonic stage of the attack. The initial fall in pressure was usually, but not always, preceded by a slight plantar flexion of the foot. It was regularly preceded by a somewhat deeper inspiration than before, and the succeeding sharp rise was contemporaneous with a very deep expiration, which heralded the onset of the tonic stage of chest movement. This relationship is clearly shown in fig. 14. In all attacks the tonic chest movement subsided more rapidly than the tonic stage in the limbs. Where the movement of the arm was recorded, as in fig. 14, it was found that the clonic stage ceased earlier in this member than in the lower limb. Particular care was taken to protect the decompression opening in the skull from any adventitious pressure. The cerebrospinal fluid pressure returned to its previous level only some 30 to 60 seconds after the termination of the attack. It will be noticed that the delay in the final

PLATE III.

DESCRIPTION OF PLATE III

Plate III. Fig. 13 (Case IV).—The scale of tension recorded by C.S.F. 1 is shown at the commencement of fig. 15. The dark tracing Dfl. moves upward with dorsiflexion of the left foot.

Fig. 14.—As fig. 13, with the addition of a manometer (Arm) which by its downward deflection shows the occurrence of clasping of the left hand. Though by observation the attack appeared to commence at the same moment in foot, hand and face, this record reveals some delay in the movement of the hand.

Fig. 15 (Case IV).—As fig. 13. To show a more brief attack, with less initial rise of cerebrospinal fluid pressure.

Fig. 16 (Case V).—Tension of cerebrospinal fluid as in fig. 17. To show by more rapid recording the relationship between the individual clonic movement in face, arm, thorax and foot. Time intervals in one-fifth second are seen as vertical white strokes at the top of the record. The uppermost tracing Dfl. shows downward movement for dorsiflexion of the left foot. Below it (Resp.) movements of the thorax are recorded with downward displacement for inspiration. Below the C.S.F. pressure tracing (C.S.F.) two superimposed lines show movements of the face and arm. The darker (Arm) shows clasping of the hand; the lighter (Face), which makes only small deflections, records contraction of the left corner of the mouth.

Fig. 17 (Case V).—Shows the commencement of a generalized myoclonic convulsion following stimulation of the patient during sleep. The figure begins with high C.S.F. pressure during sleep. At the signal recorded in the upper white line at *a* the dorsal aspect of the left forearm was pricked once, at *b* once, at *c* 10 times in succession, again at *d*, and 12 times at *e*.

XUM

fall of pressure is often related to a similar delay in the return of the chest wall to the depth of full expiration.

The effect of various stimuli which appeared to be provocative of attacks was recorded. Friction of the left hand and stimulation of the sole of the left foot were tried on many occasions, on most of which they were without effect, and when they were succeeded by an attack the time of the attack was within the limits of natural variation of the intervals above noted.

Case V. Congenital syphilis. Rapid development of dementia with involuntary twitching, the latter improved by treatment. Recurrence six months later with right-sided epileptic seizures and rapid onset of right hemiplegia. Subsequent onset of left-sided myoclonic convulsions.

W. S., age 19 years, male, admitted under the care of Dr. Hinds Howell in February, 1934. He was born at full time of apparently healthy parents who had previously had three healthy children and one miscarriage. At the age of five years it was discovered that the right eye was blind and the vision of the left eye impaired. When eight years old he attended a school for the blind and made satisfactory progress. At the beginning of 1933 he was noticed to make purposeless movements of his tongue, and he had difficulty in speaking, especially when excited. In May, 1933, unsteadiness in walking and purposeless movements of his hands and legs were noticed. In July, 1933, he was admitted to hospital. His speech was almost unintelligible owing to dysarthria and he showed an advanced degree of mental impairment. In the right eye there was no perception of light : the visual acuity of the left eye was 6/36 corrected. The right eye was small with a band of corneal degeneration, probably congenital. The left eye showed some synechiæ from old iritis and a widespread syphilitic choroidoretinitis. There was constant nystagmus of moderate rate and equal components, both at rest and on lateral deviation of the eyes. The left pupil reacted to direct light. Convergence was poor. The tendon reflexes were exaggerated and he showed irregular choreiform movements of the limbs and constant grimacing. The cerebrospinal fluid showed 42 cells, total protein 0·060 per cent., globulin positive, Lange 4544332100. Wassermann reaction full positive in cerebrospinal fluid and blood. After a course of intravenous therapy speech was a little clearer and the involuntary movements were less pronounced.

In November, 1933, a left hemiplegia occurred suddenly, with complete recovery during the ensuing night. He then remained well until the end of January, 1934, when for a period of 24 hours he developed extreme restlessness of both upper limbs and trembling of the lower limbs. Three days later he again had difficulty in moving his legs and after two further days, on February 4, at frequent intervals, for 24 hours, the right arm and leg commenced a series of spasms each lasting about 10 minutes, with the gradual development of a complete right-sided hemiplegia followed by cessation of twitching. At that time, on the morning of admission, the left corner of the mouth and the left hand commenced to twitch and there was occasional vesical incontinence. He was admitted to hospital in a stuporous condition. Attacks of twitching were observed in the left corner of the mouth and left eyelids ; more rarely there was twitching of the jaw. The left hand was rarely quiescent, displaying purposive movements, and in addition irregular myoclonic movements of the fingers, or of one finger alone, and occasionally of the wrist, elbow and shoulder. All movements were much increased by interference, which promoted a directed resistance, and ceased during periods of quietude. Occasionally the left thigh flexed or abducted and the

great toe dorsiflexed. The left upper and lower limbs were of normal tone and no defect of muscular power was demonstrable. Reflex grasping could be elicited by stimulation of the palm of the left hand. The right side was completely paralysed and immobile, with increased flexor tone in the upper limb and extensor spasticity in the lower limb. The tendon reflexes were present and equal in the upper limbs, brisk and equal in the lower limbs, the abdominal reflexes were absent on the right side, and both plantar responses were extensor. Cooperation for sensory examination was not possible, but pin-prick evoked more general reaction from the left side than from the right. The cerebrospinal fluid was reported to contain 23 cells per c.mm., total protein 0·037 per cent., globulin weakly positive, Lange 1223442210. Wassermann reaction full positive in cerebrospinal fluid and blood.

On February 6 four drams of paraldehyde per rectum reduced the involuntary movements to occasional clonic twitches of the left side of the face and the left hand, which still occurred in greater number, together with resistive movements of the left arm when the patient was disturbed. His temperature varied between 100·2° and 103° F., his pulse between 108 and 138. Minor attacks continued in spite of three further administrations of four drams of paraldehyde, until his death two days after admission. A postmortem examination disclosed considerable dilation of all cerebral ventricles due to granular ependymitis, with chronic arachnoiditis, and accompanied by considerable cortical degeneration (dementia paralytica).

Analysis of attacks recorded on February 5, 1934.—In this patient the cerebrospinal fluid pressure was found to be undergoing wide fluctuations during the intervals of sleep or stupor between the attacks (figs. 18–21). There was no accompanying fluctuation of respiration. The maximum pressure was 300 mm., with falls to 160 mm., lasting 40 seconds, as at *a* in fig. 18, with considerable fluctuation in extent and duration. Before the onset of an attack the average pressure would fall gradually and slowly to about 150 mm. Rapid irregular movements of the face, of the foot and of the chest would commence concurrently and increase in irregularity, the pressure at first showing only fine rapid fluctuations corresponding to the movements of the chest. After an interval of some 30 to 40 seconds the movements of the chest would become more violent and the pressure rise slightly. About this time rapid irregular clonic movement commonly commenced in the upper limb. The subsidence of the attack usually began with subsidence of the facial movements, but isolated irregular twitching of face, arm, trunk and leg continued to occur for many seconds, with sometimes an irregular fresh outburst in the arm or trunk, as in figs. 19 and 20. With the subsidence of twitching the pressure at first fell to the region of 100 mm. of water for an interval and rose in a series of increasing fluctuations to reach its original high level. The patient was at this time noted to be returning to his original sleeping state from which the attack had awakened him.

Rapidly moving records of the twitching during an attack showed the complete independence of the myoclonic twitching in the face, arm, trunk and leg (fig. 16). The series of events recorded in figs. 18–21 was repeated in two other complete records of attacks, differing only in the relative violence and duration of movement. Occasionally a few myoclonic twitches of the arm

XUM

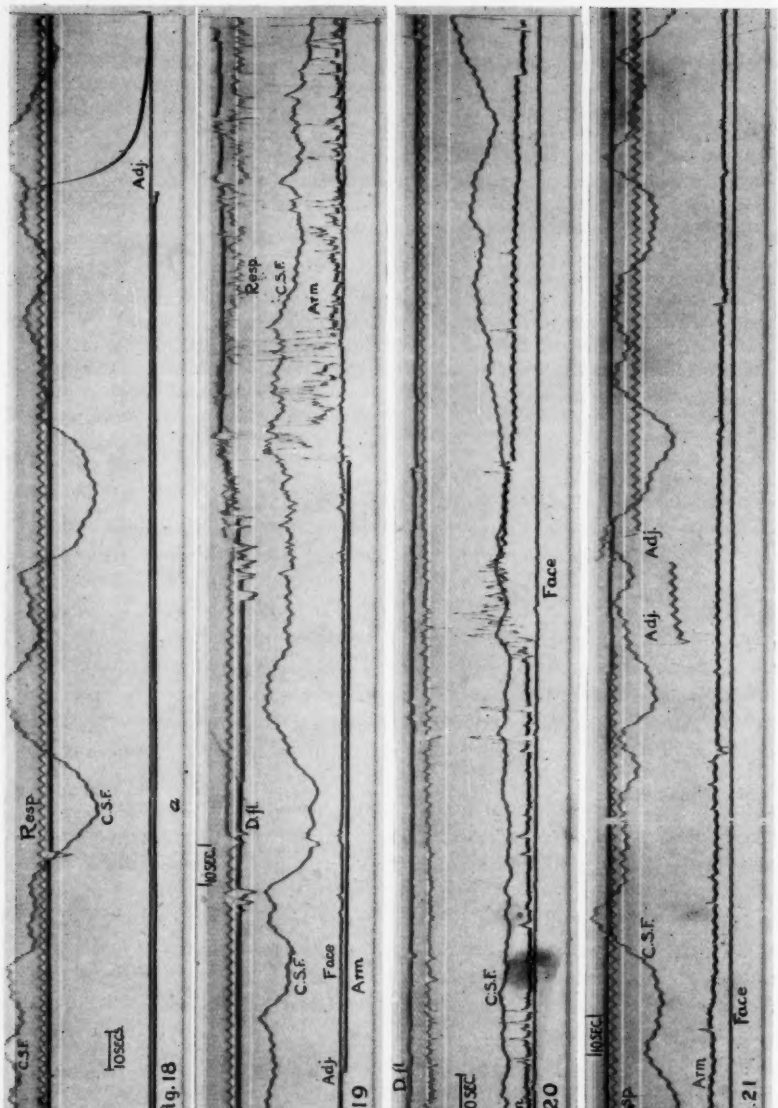

300
250
200
150
100
50
MM
C.S.F
Resp
C.S.F
10SEC
Adj
Fig.18
a
10SEC
D.ft
Resp
C.S.F
Arm
C.S.F
Adj
Face
Arm
Fig.19
D.ft
Resp
10SEC
C.S.F
Arm
Face
Fig.20
D.ft
10SEC
Resp
C.S.F
Adj
Adj
Arm
Face
Fig.21

PLATE IV.

appeared spontaneously in the trough of some of the larger fluctuations in the drowsy state. Such a ' minimal fit ' is shown in fig. 22. During the stuporose state stimulation, for example, by pricking the forearm, resulted in the gradual awakening of the patient with fall of the cerebrospinal fluid pressure in a series of wide fluctuations. When the pressure was low myoclonic twitching would commence abruptly, as in the spontaneous attack (fig. 17).

A loud clap of the hand during the drowsy state did not provoke myoclonus, although almost any stimulus increased the myoclonus when present. Inhalation of amyl nitrite during the height of an attack did not modify the cerebrospinal fluid pressure and affected the movements only in that they were slightly more violent for a brief interval. The rise of cerebrospinal fluid pressure during a fit appeared to be directly related only to movements of the chest and upper limb. Removal of 6 c.c. of cerebrospinal fluid did not modify the course of the attack. At the moment of removal the pressure was 240 mm., the previous fluctuations being small and reaching to 200 mm., the pressure after removal was 140 mm., and rose after 20 seconds to 200 mm., falling to 180 mm. After removal of another 6 c.c. the pressure remained steady for 30 seconds at 100 mm.

Case VI. Generalized convulsions at long intervals for seven years. For three years more frequent left-sided attacks, commencing in the left lower limb, without loss of consciousness. Continuous twitching of left foot for three months, with occasional spread to whole left side. Slight spastic weakness left lower limb. No organic cause found on cerebral exploration.

E. S., age 43 years, female, admitted under the care of Dr. Adie in March, 1934. No family history of epilepsy. Previous health good. Eleven years earlier, three months after the birth of her last child, she had a mild febrile illness during which there was an attack of unconsciousness with incontinence of urine. Twelve months later she again had an attack of unconsciousness with incontinence of urine. She was unable to say whether she had a generalized convulsion. From that time until three years before admission at long intervals similar attacks occurred. In one of these the tongue was bitten. In the last three years the attacks were more frequent, and more especially related to menstruation. In these without warning the toes of her left foot would become plantar-flexed, the left lower limb would be drawn up in a jerky manner, the left side of the abdomen and chest would become involved, then the upper extremity

PLATE IV.

DESCRIPTION OF PLATE IV

Plate IV.—Figs. 18, 19, 20 and 21 (Case V) form one continuous record of the transition of events from sound sleep at the commencement of fig. 18, to drowsiness at the commencement of fig. 19, a prolonged myoclonic convulsion in the latter half of fig. 19 and the first part of fig. 20, and finally to sleep in the latter half of fig. 21. The cerebrospinal fluid tension (C.S.F.) for all figures is shown at the commencement of fig. 18. Movements of the foot, face and arm are shown by D.fl., Face and Arm as in fig. 16. At Adj. at the end of fig. 18 and the commencement of fig. 19 the tension of the arm balloon was adjusted but without disturbance of the patient, as was also the respiratory recorder at Adj. in fig. 21.

XUM

3. Herpes zoster oticus with involvement of both facial and acoustic nerves.

4. Herpes zoster of the occipital region with facial paralysis, hyperacousis and tinnitus.

5. Herpes zoster of the occipital region with involvement of facial and acoustic nerves.

6. Herpes zoster affecting the ear or adjacent region of the head with disturbance of the acoustic nerve but no facial paresis.

The sixth nerve is rarely involved in association with herpes of the ear region. In a few cases, the facial or the acoustic nerve was affected when the herpes occurred elsewhere on the body.

The general health may be affected in all cases of this type. Leucocytosis has also been described and occasionally an increase in both cells and protein content of the cerebrospinal fluid. The herpetic eruption is found in the area supplied by the fifth nerve in about 35 per cent. of the cases; in others it occurs in the areas of the seventh, ninth or tenth nerves. Sometimes it may arise simultaneously in the areas supplied by several nerves.

The author of this communication considers that the lesions of the facial and acoustic nerves are the result of herpetic infection of their ganglia. Occasionally herpetic infection of these nerves may occur without any eruption. Such cases have been recorded by Frankl-Hochwart under the heading of 'Polyneuritis cerebralis meniereformis.'

It is noteworthy that the author recommends that a herpetic eruption of the tympanic membrane should not be interfered with provided there is not much pain, as the opening of the middle ear may give rise to a secondary infection.

M.

[22] **The present conceptions of herpes** (Les conceptions actuelles de l'herpes).—M. Albeauz-Fernet and P. Collart. *Gaz. des hôp.*, 1933, **106**, 1467.

In 1904 Ravaut and Danet by means of lumbar puncture showed the constancy of nervous involvement in genital herpes and especially in recurrent herpes. From 1910 onwards the disease was considered an inoculable infectious disease. It is now generally admitted that herpes is a specific disease of which the cutaneous lesions represent only one manifestation. Multiple causes favour the appearance of the eruption, the most frequent being certain infectious diseases, cerebrospinal meningitis, pneumonia, spirochætosis icterohæmorrhagica, local infections, intoxications, endocrine disturbances, etc. These, however, are only occasional causes which, by the disturbances they bring about, provoke the hitherto latent virus. Recent work has shown that an invisible and filtrable virus is concerned. Levaditi considers it is an ultrafiltrant virus. The authors of this paper give in detail

the results obtained in the course of their experiences and describe the pathological anatomy and the clinical aspects of the affection which vary considerably according to the localization. As regards diagnosis, they stress the difficulty when the herpes lies at the level of the genital organs and particularly the confusion which may arise between herpes with confluent vesicles and a syphilitic chancre. Finally, they note that a meningitis may occur although the proof of its existence as a clinical entity is difficult to establish, the cerebrospinal fluid changes being very transient. According to Sendrail, a positive inoculation of the cerebrospinal fluid in the cornea of a rabbit would be of no definite value; it would confirm the presence of the virus in the organism but not its pathogenic rôle. An important bibliography is appended.

C. W. D.

[23] **Myotonia atrophica with cataract.**—Leo L. Mayer and Joseph A. Lucham. *Arch. Neurol. and Psychiat.*, 1933, **30**, 810.

The authors review the literature and submit a report of three additional cases The principal features of the disease can be summarized as follows:—

Myotonia atrophica (myotonic dystrophy) is a distinct nosological entity characterized by: (1) myotonia, in the active form usually limited to the hand grasps; (2) muscular atrophy of a selective distribution; and (3) dystrophic extramuscular phenomena, chief among which is a distinctive form of presenile cataract. The disease is probably heredofamilial, as the studies of Fleischer indicate. It usually begins between the ages of 18 and 35 and predominantly affects males.

Active myotonia is ordinarily limited to the fist-closing muscles, but sometimes involves the tongue and other groups of muscles. Mechanical myotonia, shown by a persistent dimple or furrow at the point of impact when a muscle is percussed, is most commonly found in the thenar and hypothenar muscles and in the deltoids. The myotonic electrical reactions are often influenced by the presence of atrophy in the muscles examined. All three forms of myotonia are not usually found in the same muscle. Myotonia tends to appear early, before the development of atrophy, and may disappear with the advance of the atrophy.

The muscular atrophy is selective, involving the facial muscles, the sternocleidomastoids, the muscles of the forearms, the vasti of the thighs and the dorsiflexors of the feet. The hatchet-like physiognomy, combined with ribbon-like sternocleidomastoids and wasting of the muscles of the forearms, gives rise to a strikingly characteristic picture.

The extramuscular symptoms include cataract—essentially a presenile cataract—revealed on gross examination in about one-half of the published cases. However, cataract will probably be found to be one of the most nearly constant findings in this condition if the lenses are examined by means

of a slit lamp. Early, the appearance of fine, dust-like opacities under the anterior and posterior capsules is characteristic of this form of cataract; later, starlike formations may be found. Cataract is of great significance in the hereditary transmission of the disease, since members of affected families may present cataract alone (without muscular symptoms).

Testicular atrophy, monotonous nasal speech, acrocyanosis, premature baldness, increased secretion of tears and loss of tendon reflexes even in healthy-appearing muscles are dystrophic phenomena that are encountered with varying frequency in myotonia atrophica.

The pathological basis for the atrophy appears to have been found within the muscles themselves (rather than in their related motor cells within the spinal cord). There is some histological evidence to suggest that the vegetative nervous system within the central neuraxis may be pathologically implicated as the cause of the myotonia. The occurrence of cataract and of tetanic muscular phenomena in states of parathyroid deficiency has led investigators to search for evidences of parathyroid insufficiency in myotonia atrophica. In this regard, certain recent observers have stressed a diminished calcium content of the blood, signs of latent tetany and the cataract itself as indicating that parathyroid insufficiency is involved in the pathogenesis of myotonia atrophica.

R. M. S.

[24] **Posttraumatic paresis of the posterior cord of the brachial plexus** (Posttraumatische Paresen im Bereich des hinteren Bündels des Plexus brachialis).—J. HEMPEL. *Münch. med. Woch.*, 1934, **81**, 93.

THREE cases are recorded in which four to six months after a comparatively slight trauma of the shoulder region there was found weakness in the external rotation of the arm at the shoulder and in raising the arm forwards. The muscles over the scapula were wasted; in one case there was also slight wasting of the deltoid and in another winging of the scapula. In two of the cases cutaneous sensation was normal and the third showed diminished sensibility over the lower part of the scapula. In all three cases the electrical reactions were impaired in the supraspinatus and infraspinatus muscles. The electrical change found in the supraspinatus is remarkable as the trapezius was not apparently affected. The above symptoms are described as a consequence of a lesion of the suprascapular nerve which usually arises as a special branch from the outer cord of the brachial plexus, but in some individuals this nerve arises from the circumflex. The suprascapular nerve also supplies the teres minor and the upper part of the serratus magnus; consequently in some cases external rotation of the arm may be seriously impaired and there may also be some dislocation of the inner border of the scapula.

M.

PROGNOSIS AND TREATMENT

[25] **The treatment of juvenile general paralysis.**—Howard W. Potter. *Psychiatric Quarterly*, 1933, **7**, 593.

Of 60 cases of juvenile general paralysis 38 were treated with malaria or tryparsamide or both, 20 with common arsenicals and bismuth or mercury, and two with radiothermy and diathermy respectively. Of the 20 treated with common arsenicals, 17 continued to undergo a mental and physical deterioration and several of these died. In 27 of the 38 cases treated with malaria or tryparsamide the treatment was effective in so far as it prevented further deterioration; five of these gained a complete remission and nine a partial remission. In the great majority of instances a period of from two to five years had elapsed after the beginning of the treatment. In studying the factors that affect the prognosis in the 38 cases treated with malaria or tryparsamide the prognosis was found to be better in (*a*) patients who prior to the onset of the paresis were of normal mental level; (*b*) patients who were in or past adolescence when the symptoms developed; (*c*) those showing the expansive and confused reaction types; (*d*) those in whom the elapsed time between the onset of the disease and the treatment did not exceed two years. Sex, character of the onset and antiluetic treatment prior to onset did not appear to affect the prognosis favourably or unfavourably.

C. S. R.

[26] **Treatment of dementia paralytica with typhoid H. antigen vaccine.** —M. T. Schnitker. *Arch. Neurol. and Psychiat.*, 1934, **31**, 579.

Twenty-five patients with dementia paralytica, physically unsuitable for treatment with malaria, whole typhoid vaccine or diathermy, were treated with typhoid H antigen (flagellar) vaccine alone and in combination with tryparsamide.

In the author's opinion the symptomatic and serological improvement obtained by this method compares favourably with results obtained with other forms of fever therapy.

Typhoid H antigen vaccine is indicated for persons who cannot withstand the rigors of the other heroic forms of therapy because (*a*) although the hyperpyrexia equals that obtained with whole typhoid vaccine, the concomitant illness is far less severe; (*b*) it does not involve superimposing a second (and perhaps lethal) infection on an already existing one, as with malaria; (*c*) it does not involve the risk of burns and renal damage, as with diathermy; (*d*) its contraindications are but four, namely, (1) severe cardiorenal disease, (2) active pulmonary disease, (3) severe cachexia, and

(4) acute infection with a rapid sedimentation rate; (*e*) its administration and control are simple, and its use in the hands of the general practitioner, both in the hospital and in the home, is safe; (*f*) in Schnitker's hands it has not been associated with complications or sequelæ.

Improvement was found not to be directly proportional to the height of the temperature obtained, but seemed to be related to the total number of hours of fever. Better clinical results were obtained by combining the administration of tryparsamide with the fever treatment, the injections being given at the height of the fever.

R. M. S.

[27] **Studies in endocrine therapy in epilepsy.**—Calvert Stein. *Amer. Jour. Psychiat.*, 1934, **13**, 739.

The results of therapy with a selected group of endocrine preparations on 68 epileptics, one narcoleptic, and 75 non-epileptic controls of both sexes are given. None of the preparations that were used was specific for convulsions—neither curing them in the epileptics, nor causing them in the controls. There was some evidence, however, that in a few cases overstimulation of the ovaries might be indirectly provocative of convulsions. The majority of those that received thyroid substance, either alone or in combination with other glands, showed definite clinical improvement in metabolism, bowel activity, and general wellbeing. Blood-pressures tended to be subnormal and there was some evidence that dried suprarenal gland by mouth, as well as thyroid, will raise the pressure from 10 to 30 mm. and maintain the rise as long as medication is continued. The best results in both epileptics and controls were obtained with pituitary, thyroid and suprarenal, and pluriglandular therapy was more efficacious than single therapy except in treatment with thyroid. Endocrine therapy should be but one part of a general therapeutic programme, for there is no known endocrine disorder that appears to be specifically responsible for the production of epileptic seizures. Patients with obesity and constipation offer the best prospects for improvement with oral administration of thyroid and whole pituitary in combination. Those who improved in general health while under treatment did not show any reduction in the frequency or severity of their fits, but did show a lessening of postconvulsive debility, headache and weakness, and also a marked shortening of the post-convulsive period of convalescence. It is reasonable to expect that if the clinical improvement in metabolism, gastrointestinal function, and general wellbeing of epileptics can be maintained for a period of several years at least, the convulsive threshold may also rise to the point of overcoming their seizures.

C. S. R.

Psychopathology

PSYCHOLOGY

[28] **A statistical study of dreams in relation to emotional instability.**—F. K. Berrien. *Jour. Abn. Soc. Psychol.*, 1933, **28**, 194.

Students taking introductory courses in psychology were experimentally investigated in order to throw light on the question—Do emotionally unstable individuals recall more dreams than their relatively stable associates? It was concluded that something besides emotional instability was operative in determining the frequency of dreams. The evidence here set forth gives no foundation for the Freudian concept of dreams and must be looked upon as putting the analysts on the defensive when they begin talking about the relationship between dreams and emotional instability. On the contrary, no evidence appears here to question the assumption of H. J. Watt, who says the chief difference between the thinking of daily life and of dreams must surely lie in the initial source of each. In sleep we start usually, or after the slightest reflection from within or without the body, with favoured but unsatisfied or unrealized attitudes of mental action. These are the parts of our mind that have the most latent energy and are least asleep.

C. S. R.

NEUROSES AND PSYCHONEUROSES

[29] **Psychoneuroses of childhood: their ætiology and treatment.**—Melitta Schlideberg. *Brit. Jour. Med. Psychol.*, 1933, **13**, 313.

The neurotic symptoms of childhood are by no means due solely to wrong behaviour on the part of the parents; they occur also under excellent environmental conditions. They are caused by conflicts which are at work in every child. These can be increased or diminished through environmental influence but can in no case be avoided completely. Such symptoms sometimes appear in the first weeks of life. Some infants from the very beginning show anxiety and sometimes as early as the second or third month of life this anxiety takes on the form of a definite phobia. There are others who from the earliest age suffer from conversion symptoms, vomiting, constipation, or fits. Many children develop anxiety in the second or third year, often of psychotic character. 'Naughtiness,' lying and stealing as well are mostly related to neurotic conflicts. Asocial behaviour and abnormal character development often start in early childhood. Frequently enough such symptoms disappear in later life, but this often merely indicates that the conflict is finding no outlet—a psychic situation which may be unfavourable for the child's development. Disappearance or diminution of neurotic symptoms in the latency period is caused by strengthening of the ego and

weakening of instinctual urges. The child attempts to suppress or conceal its conflicts, forbidden instinctual desires and troublesome neurotic symptoms, and in doing so is often strongly influenced by its environment. Mental balance acquired in the latency period through suppression of instinctual energies is upset by the surge of reinforced instinct at puberty. Outbreaks of psychosis or neurosis often occur at puberty and abnormal character development is usually recognized at this age.

C. S. R.

PSYCHOSES

[30] **Clinical variables in schizoid personalities.**—Jacob Kasanin and Zitha A. Rosen. *Arch. Neurol. and Psychiat.*, 1933, **30**, 538.

The aim of this study was to answer two questions: (*a*) Do patients with similar schizoid personalities have similar clinical pictures when they break down? (*b*) Can the schizoid personality be described in terms of a given constellation of traits? This type of personality is defined as possessing the following five characteristics: few friends, shyness, seclusiveness, closed-mouthedness, and extreme sensitivity. Only 33 of 327 patients were found to have this combination of traits in prepsychotic life; 24 of these had schizophrenia out of a total of 151 with schizophrenia. The clinical pictures in these cases varied greatly from case to case with only slight superficial resemblances. In spite of the alleged unfavourable constellation of traits in the personality, the recovery rate was higher than in an unselected group of schizophrenic patients or in general hospital populations. Analysis of factors favouring recovery did not reveal any single especially significant condition in all cases. It seems that certain conditions in present-day culture lead to the hospitalization of maladjusted males at an earlier age than females. Only 15.9 per cent. had the specific combination of traits delineating the schizoid personality. Significantly associated with the nucleus of five traits were 'little self-assertiveness,' 'no sense of humour,' 'frequent day-dreaming,' 'little output of energy,' 'feeling of inferiority,' 'fusses over pain,' 'very absent-minded,' 'neurotic traits in childhood,' and 'unusual attachments' to some member of the family in adult life. In the transition from childhood to adult life, the neurotic traits decreased while all the schizoid traits increased. A review of various clinical data reveals that the most frequent factors associated with the schizoid personality were an over-attachment to the family, maternal over-protection, paternal neglect, physical defects and anomalies present in practically all of the cases, and unsatisfactory heterosexual adjustments. The description of the schizoid personality by traits does not give a true picture of the personality or of its dynamic relation to the psychosis.

C. S. R.

[31] **The experiential aspects of dementia præcox.**—Anton T. Boisen. *Amer. Jour. Psychiat.*, 1933, **13**, 543.

This study proceeds from the hypothesis that dementia præcox has to do with the urge for self-realization and that it consists of two chief types. One has malignant tendencies. Here the urge for self-realization has either become short-circuited as a result of the closing of the configurations on a level which leaves the individual isolated from his fellows and shut off from his own objectives. In the other there is generally the awareness of personal failure and inability to face the inner bar of judgment. Thus the terrified individual becomes preoccupied with a narrow circle of ideas and passes into an abnormal condition. Though an illustrative case is given to serve as a base-line for an examination of the inner meaning of the schizophrenic experience, the findings here are founded upon a study of 176 cases. In general these were found to have more than their share of hereditary taint; more than their share of unfavourable environmental influences; and while seldom feebleminded they were somewhat below par intellectually. Physically they were a trifle undersized and had less drive and vigour than normal. Somewhat more subject to physical disease, about two-thirds had more or less endocrine malfunction. But in all this there was nothing distinctive. In 36 per cent. there were clear maladjustments in the fields of sex, vocation, and society, and in 75 per cent. the same could be said of two of the fields.

Following the idea that the primary evil in dementia præcox involves the sense of personal failure, isolation and an intolerable loss of self-respect result. Three methods of dealing with this sense of personal failure were traced —*drifting* into mental refuges, the dream world becomes the real world. Through refusing to admit error or defeat, others resort to *delusional misinterpretation*. Few escape becoming aware of their danger with more or less emotional disturbance, and in extreme forms *panic* may ensue. These general reaction types usually occur in combinations.

In 55 of the cases the onset was sudden and involved acute emotional disturbance; in 69 it was of the subacute type; and in 52 it was insidious. Only in seven cases were there any factors in the immediate life situation which might account for the mental disturbance. The sense of failure may be recognized, concealed, or evaded. Out of the 176 cases, 130 showed manifestations of concealment. This is found in four chief forms, the most frequent being the *externalisation of conscience* (113 cases), as seen in accusing voices, the mind being read, ideas of being poisoned or killed. *Transfer of blame* as noted in paranoid ideas of electrical currents, or being superpersonally influenced was evident in 70 cases. *Fictitious self-importance* appeared in a large number, and in 10 was sufficiently powerful to effect a comfortable delusional reconstruction. Lastly, the *incapacitation* reaction in which physical illness is made to bear the responsibility for the personal

failure was noted in 29 cases. The chances of recovery depend largely on the possibility of making an adjustment in the life situation which will be acceptable to the patient. Where the situation is lacking in constructive factors and admits of no solution, the patient may be restored temporarily but is not likely to remain well. The significance of the reaction patterns must be carefully considered, and the malignant tendencies must be distinguished from the acute disturbance characterized by ideas of death, of cosmic catastrophe and cosmic identification. The more sudden the onset and the more acute the disturbance, the more likely is the patient to recover. Suffering may be looked upon as remedial. The presence of inappropriate moods and attitudes is a bad sign. Attitudes of frankness and self-blame are favourable indications. 'Voices' indicate a stirring of the deeper mental levels, something which in itself may be helpful as well as destructive. Their chief significance lies in what they reveal as to the inner trends and attitudes.

C. S. R.

[32] **Order of birth in manic-depressive reaction.**—Harold H. Berman. *Psychiatric Quarterly*, 1933, **7**, 430.

Out of 100 cases of manic-depressive psychoses 48 were found to be among the first-born. Twenty-two cases were among the last born. The reaction to the birth of a younger member in a family frequently appeared to be a predisposing factor. Early childhood disorders should be treated as manic-depressive reactions in the hope of averting frank attacks of psychosis in later life.

C. S. R.

[33] **The blood cholesterol in schizophrenia.**—Joseph M. Looney and Hazel M. Childs. *Arch. Neurol. and Psychiat.*, 1933, **30**, 567.

Approximately 50 men with schizophrenia were studied at intervals for seven months. The cholesterol content was determined by the method of Myers and Wardell. The mean values were: 146 $\pm$ 3 mg. per 100 c.c. for the first period, 161 $\pm$ 2.8 mg. for the second period, and 166 $\pm$ 2.5 mg. for the third period. The mean value for 26 normal men was 175 $\pm$ 5.2 mg. Both the schizophrenic patients and the controls showed great variation in the cholesterol values, the former having a standard deviation of about 20 mg., and the latter of 27 mg. The difference between the mean value for the first period and those for the last two periods is believed to be due to a seasonal valuation in the cholesterol content of the blood. No correlations could be shown between the blood cholesterol and the basal metabolic rate or the emotional status. Schizophrenia seems to be characterized by a slight degree of depression of the cholesterol content of the blood.

C. S. R.

[34] **The fæcal flora in catatonic dementia præcox.**—Barbara McGinn, Mary E. Raney, and Nicholas Kopeloff. *Psychiatric Quarterly*, 1933, **7**, 260.

Bacteriological examination of the fæcal flora of four catatonic dementia præcox patients was repeated monthly for four months. The hydrogen ion concentration, total bacterial count, predominant aerobes and anaerobes, all fell within the limits of variation with the normal. The incidence of *B. acidophilus* was less than normal, but the character of the flora was fermentative rather than putrefactive. No unusual microbe was isolated with sufficient consistency to indicate that the bacterial flora might be causally related to the mental disease process.

C. S. R.

PSYCHOPATHOLOGY

[35] **An analytical review of a series of cases of insanity with pregnancy.**—C. B. Bamford. *Jour. of Ment. Sci.*, 1934, **80**, 58.

In this review the old conception that these cases offer a favourable prognosis from the point of view of complete recovery of the mother is by no means confirmed. At Rainhill Hospital insanity with pregnancy (taking no account of puerperal and lactational cases) was found to be comparatively rare, accounting for less than 10 per cent. of total admissions. Ninety-seven cases are here examined. In only 17 cases was there evidence of any psychopathic inheritance, and of these only six recovered. In over 25 per cent. it seemed that some associated disease brought an extra strain to bear and was responsible for the breakdown. Over 75 per cent. were admitted beyond the seventh month, and it would appear that the second half of pregnancy is the time fraught with risks of serious mental disturbance, particularly the seventh or eighth month. The absence of obstetrical difficulties and complications seems worthy of mention. The puerperium, too, was especially free from infective processes. The conclusion was arrived at that a large number of cases were potential psychopaths and would have become insane sooner or later quite independent of pregnancy. Amongst the unrecovered cases (59) there were 12 mental defective, 13 epileptics, seven general paralytics, 12 with dementia præcox, 11 with manic-depressive insanity, two with delusional psychoses, and two with alcoholic dementia. The problem of the correct diagnosis of the cases that recovered exercised the mind of the writer for some time. Most were regarded as confusional (for want of a better title) or manic-depressive and a few had mixed symptoms which did not fit suitably into either category. Nevertheless, the confusional cases did not conform to the usual type. There was scarcely any associated bodily disturbance of any severity and very little reliable evidence of any severe degree of bodily intoxication. With the exception of three cases which improved before

delivery most failed to show any improvement until the second month. For a time after delivery there was an acute exacerbation of the mental symptoms. In six of the cases improvement was deferred until six months had passed, but they then made a full recovery. In the average case, recovery may reasonably be expected about the third month following confinement, and this would make the probable duration of such a psychosis as six months. It is to be noted that in no case was premature labour artificially brought about, and it is thought that there must be very few cases of insanity which either in the maternal or fœtal interest require such a step.

C. S. R.

[36] **The nature of emotion and its relation to antisocial behaviour.**—Fred Brown. *Jour. Abn. Soc. Psychol.*, 1934, **28**, 446.

From this study it is concluded that the ' psychopathic personality ' is not to be attributed to an organic or functional defect. Psychopathic behaviour or emotional maladjustment is strongly indicative of trial-and-error attempts to utilize the constantly accumulating energy generated in the presence of a stress-strain situation for which the individual possesses no adequate outlet. Consequently, otherwise normal reactions become overcharged with energy and result in antisocial modes of response or else less adequate responses are utilized to relieve the tension state. The fault lies in the *type of behaviour* utilized rather than in any fundamental physio-organic disorder. Since all social behaviour is learned, there is great hope for the ultimate elimination and prevention of antisocial modes of conduct now designated as ' emotional ' in nature.

C. S. R.

[37] **Some observations on lipoid metabolism in mental disorders.**—J. S. Sharpe. *Jour. of Ment. Sci.*, 1934, **80**, 75.

The result of these investigations shows that the lecithin content of the blood in mental disorder exhibits no important variation from normal. The cholesterol content of the blood in early dementia præcox is decreased by about 25 per cent. Later there is an increase by about 30 per cent., as chronicity advances, and it remains at that figure. This may be due to a hyperactivity of the suprarenal glands. The coefficient of utilization is low. In melancholia and confusional insanity the blood cholesterol is slightly on the high side, but the variations are small. The coefficient of utilization is very low in these groups, denoting deficient oxidation and low metabolic activity. Recurrent mania cases show a very high blood cholesterol, particularly during an acute attack. This cyclic nature of the condition seems to suggest some derangement in metabolic activity, probably centred round the suprarenal glands, as evidenced by the blood cholesterol increase. Further, the coeffi-

cient of utilization is very high, indicating increased metabolic rate. There exists in the blood in certain melancholic and confusional states a powerful depressor substance having a choline-like action on the isolated frog heart. This substance is antagonized by adrenaline. Normally, the depressor substance is in such small concentration as to be almost undetectable. In dementia præcox and recurrent mania this substance exists in very small quantities, approaching the normal figure as determined by the physiological test. Evidence points to a deficiency or absence of choline in melancholia and confusional insanity. This substance may be conveniently termed 'cholinase.' The clinical feature of these cases is that of hypotonia.

C. S. R.

[38] **Studies of blood-sugar curves in mental disorders.**—S. KATZENELBOGEN and E. FRIEDMAN-BUCHMAN. *Amer. Jour. Psychiat.*, 1933, **13**, 321.

THE carbohydrate metabolism, as tested by the blood-sugar curves, was studied in 116 psychotic patients. These included 31 cases of psychoneurosis, 50 cases of manic-depressive insanity (out of which six were manic), 20 of schizophrenia, and seven cases of organic psychosis. The curves were evaluated, first, by the intensity of the alimentary hyperglycæmia (following the ingestion of 50 gm. of glucose); second, by the extension of the glycæmic reaction over a two-hour period (hyperglycæmic index); and third, by both the height and extension of the curve combined (hyperglycæmic area). Abnormal curves were obtained in each psychotic group; in the psychoneuroses, 64 per cent.; in mild manic-depressives, 72.9 per cent.; in the schizophrenics 60.7 per cent., when the curves were evaluated by the climax of the hyperglycæmia. When estimated by the hyperglycæmic area, the values were 62.5, 79.2, and 72.7 per cent. respectively; in the organic psychoses, 100 per cent., judged by both methods of evaluation. The abnormal curves showed similar characteristics in the four reaction types. It is believed that the apparently similar disturbance of the carbohydrate metabolism in the different psychotic reaction types may be related to a common denominator, that is, affective disorder. Thus, the abnormal blood-sugar curves in these patients were considered as physiological accompaniments of psychobiological reactions.

C. S. R.

[39] **Blood and urine chemistry during the specific dynamic action of glycine in normal subjects and in schizophrenics.**—CHARLES REID. *Jour. of Ment. Sci.*, 1934, **80**, 379.

AFTER glycine the amino-N content of the blood reached its maximum near the end of the second hour. Blood urea-N increased slowly and steadily

during the period of observation. The mean increase in the seventh hour after glycine was about 4·0 mgm./100 c.c., but the actual increase in a particular case appeared to depend entirely on diuresis and the urea excreted. Blood-sugar decreased about 10-15 mgm. per 100 c.c. after glycine ingestion, but tended to approach the fasting level in the course of six to seven hours. The non-glucose-reducing substances were not significantly altered. The non-protein nitrogen fraction (non-urea and amino-N) of the blood was increased after glycine administration. Nitrogen elimination was much increased also and the excess urea-N excretion during six hours of the post-glycine period amounted to 0·23 of the nitrogen given as glycine. The total nitrogen of the urine (less the amino- and urea-N fractions) was increased after glycine, and this was due either to increased elimination or to increased production in the tissues, or to both. Sulphate excretion was maintained at a higher level than was found for controls during the postabsorptive period. It appeared that the sulphate excretion provided a more reliable index of specific dynamic action than the nitrogen excretion during the period of observation in the experiments dealt with here. The examination of schizophrenics after glycine ingestion did not reveal any striking deviation as to their blood and urine chemistry from those in normal subjects. The character of the mean blood amino-N and urine amino-N curves suggested delay in the absorption of the ingested material as compared with normal. Blood nitrogen and urine nitrogen estimations were not significantly different in schizophrenics from the normals. Approximately the excess urea-N excretion after glycine amounted to 0·21 of the nitrogen ingested as glycine. Sulphate excretion on the whole was less for schizophrenics than for the normals, and this could be due to decreased specific dynamic action in these psychotics or to a diminished absorption rate of the ingested material. Blood urea values in schizophrenics and in normal controls after the giving of 15 gm. urea in 100 c.c. were suggestive of delayed absorption, since the rise in blood urea was slower in the former. Conclusions based on blood urea values after glycine without consideration of the urea excretion are not justifiable. The attempt to demonstrate variations in the specific dynamic action of foodstuffs or glycine by ingestion methods in psychotics is also unjustifiable, in view of the variations in the processes of absorption which have been demonstrated in both normal and psychotic subjects.

C. S. R.

PROGNOSIS AND TREATMENT

[40] **Prognosis in manic-depressive psychoses.**—Reginald R. Steen. *Psychiatric Quarterly*, 1933, **7**, 419.

From a study of 493 cases it is concluded that an individual would be most likely to recover from his manic-depressive attack if he had a normal previous

personality, a clear heredity, no previous attacks, or a previous attack between his 20th and 30th birthdays, and an abrupt onset of a typical manic or depressive reaction with no delusions or hallucinations. The average duration of the writer's recoveries was 1·51 years.

C. S. R.

[41] **The treatment of narcissistic neuroses and psychoses.**—L. Pierce Clark. *Psychoanalytic Review*, 1933, **20**, 304.

While we may learn a great deal about narcissism by applying the technique of ordinary analysis, we fail to help the narcissist. Some new method must be evolved. The attitude toward the mental disease is one of 'watchful waiting,' and yet a fairly large number of cases, for all practical purposes, recover. In each individual case it seems as though some combination of fortuitous circumstances has had influence. The inference is that the therapy of 'watchful waiting' allows the ego to take its own time in gradually adapting itself to the outer world. The value of the fortuitous circumstances mentioned apparently consists in their providing to the weak ego the love and support it requires, and in their granting it the extended opportunity for slowly making ready for the changes which the instincts and reality impose. The purpose of a psychoanalytic technique would be to make such an outcome less the result of chance, more the product of enlightened effort. It may be that the relationship of analyst and patient will never assume the conditions of an ordinary transference-analysis; but through a modified technique the ego is given the chance to resume its interrupted growth at a speed of its own choosing. It can be made possible to discharge emotions without at once facing the retributions of the real world. It should be a relationship where the analyst goes with the patient rather than the reverse. Thus is promised the possibility of overcoming that primary mountain of resistance which has hitherto defeated most attempts to analyse the narcissist.

The early stages of treatment may conform largely with the pleasure-principle, but eventually as the transference becomes stronger there must be slowly offered an influence towards accepting the reality-principle. The ultimate aim of therapy would be to bring together narcissistic libido and object-libido into a united release upon real activities. It must be held that it is possible to strengthen the ego. All that is asked of the patient is that he talk about himself. The problem becomes one of allowing the analyst to be recognized as not completely attached to the patient's ego but requiring some projection of libido in order to gain narcissistic satisfactions. Once a narcissistic transference is established the procedure is very similar to that followed in the analysis of the transference neuroses. The danger may be that too much may be expected of the narcissistic patient.

C. S. R.

[42] **The treatment of dementia præcox by continuous oxygen administration in chambers and oxygen and carbon dioxide inhalations.**—L. E. HINSIE, A. L. BARACH, M. M. HARRIS, E. BRAND and R. A. MCFARLAND. *Psychiatric Quarterly*, **1934, 8, 34.**

FIVE patients resided continuously for two and a half months in an oxygen chamber at a concentration of approximately 50 per cent. oxygen. During this time they received treatments of carbon dioxide and oxygen daily. After each carbon dioxide inhalation attempts were made to establish communicability with the patients. Two of the five gained a state of remission and the clinical history of these had indicated a favourable prognosis. A second group of five patients were treated similarly except that no communicability attempts were made. None of these showed improvement. A third group of five resided in the oxygen dormitory without carbon dioxide treatment and without communicability attempts. None of these showed any improvement. A fourth group of five, living under normal atmospheric conditions, were given daily inhalations of carbon dioxide and oxygen. Here one showed clinical improvement but did not gain a remission.

The blood chemical studies showed no effect of long-continued oxygen inhalation on the urea nitrogen, uric acid or blood sugar content of venous blood. Basal metabolic determinations varied so much that no consistent conclusions could be drawn. Under various psychological tests no consistent improvement was observed in the patients who did not gain a remission. From observations made, it does not appear that oxygen and carbon dioxide treatment of catatonic dementia præcox patients is to be advocated as a general therapeutic procedure.

C. S. R.

Reviews and Notices of Books

The Human Personality. By Louis Berg, M.D. London: Williams and Norgate. 1933. Pp. 309. Price 8s. 6d.

An ambitious attempt is made in this book, written from the sociologist's point of view, to sum up our knowledge of human personality. The author reviews the conclusions, as he interprets them, reached by biology, genetics, endocrinology, psychology, criminology and psychiatry regarding the nature of man and the determinants of his conduct.

The result is disappointing. Statements which cannot be supported are numerous. The section upon 'The Glandular Basis of the Personality' has many such. For example, we are told (p. 67) that the personality may be controlled by overaction of the posterior pituitary. No evidence is given, or available, to prove this. Dr. Berg describes a type of personality as the 'status thymico-lymphaticus' and tells us how it may be 'cured.' In 1931 the Status Lymphaticus Committee declared that there was no evidence that so-called 'status thymico-lymphaticus' had any existence as a pathological entity. Many old errors are repeated and no new information given in the chapter on 'Personalities in Conflict.' Statistics contradict the author's statement that 'hysteria is the most common of all neuroses.' Few will agree that 'Charcot was the first to use hypnotism scientifically'—or, perhaps, that he ever used it scientifically.

The book was written 'in view of the acute need of well integrated studies of our sometimes bewilderingly complex life.' It fails to satisfy this 'acute need,' and we must still await 'a synthesis of modern research.'

Psychology and Psychotherapy. By William Brown, D.M., D.Sc., F.R.C.P., Wilde Reader in Mental Philosophy in the University of Oxford, etc. London: Edward Arnold. 1934. Pp. 252. Price 12s. 6d.

The previous editions of this work (first published in 1921) have been reviewed in this Journal. The third edition has been enriched by the author's wider experience; and in aim and plan it is virtually a new book. Dr. Brown, while accepting much of the systems of Freud, Jung and others, declines the label 'eclectic,' preferring to regard his position in and contribution to psychology and psychotherapy as 'synthetic.' The term is not a happy one—it suggests an artificial product. This is, however, far from being the

case. The views here expounded have stood the test of time and practical application.

The book gives a balanced survey of medical psychology and as such it can be unreservedly commended. The appendices, five in number, occupy about a fifth of the book; they will appeal chiefly to the trained psychopathologist. An excellent index is provided.

E. A. B.

THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. XV. OCTOBER, 1934 No. 58

Original Papers

OBSERVATIONS ON RECORDS OF LOCAL EPILEPTIC CONVULSIONS *

By

D. DENNY-BROWN AND E. GRAEME ROBERTSON, LONDON

PAGE

INTRODUCTION 97

METHOD 98

PROTOCOLS 98

DISCUSSION 119

The Cerebrospinal Fluid Pressure 120

The Effect of Sleep 121

'Reflex' Epilepsy 122

The Incoordination of the Epileptic Discharge 124

The Periodicity of Seizures 131

CONCLUSIONS 135

INTRODUCTION

THE varied phenomena of epilepsy and the many clinical problems with which it is associated present a perennial source of interest to the clinician. The convulsion itself presents many problems which become the more interesting the more closely they are observed, though the rapid sequence and concurrence of a number of manifestations tend to confuse clinical observation. Some of the events lend themselves to analysis by graphic record, and the following account concerns records of cerebrospinal fluid pressure, respiration, and muscular movement in six patients. Though such records fall far short of any exact quantitative measurement of muscular contraction, careful control by clinical observation allows them a measure of accuracy, and in their essential feature, namely sequence of events in time, they appear to us to be reliable evidence.

* From the National Hospital for Nervous Diseases, Queen Square, London.

METHOD

The pressure of the cerebrospinal fluid was recorded by means of a mirror tambour capsule which reflected a beam of light to a moving strip of bromide paper. The pressure was transmitted from a wide lumbar puncture needle to the glass capsule by means of thick rubber tubing of small inside diameter. A small sterile chamber intervened between the needle and the tubing, and here the cerebrospinal fluid reached a level by compressing the air within the capsule system. The capsules used are identical with those we [5] have described in detail for use in the measurement of vesical pressure, and are calibrated after each experiment by measuring the deflection produced by water columns of various heights. The periodicity of the system is well damped by the slight fluid movement; the latency is negligible at the rates of recording used. The patient lay on the least active side, on a wide couch, for periods up to three hours. Movements have been recorded by the attachment of pressure balloons over the chest wall, and over the muscle concerned, with due precaution to avoid the possibility of movement by other muscles. Changes in pressure within the balloons was transferred to capsules with mirrors which recorded on the same bromide paper. The record of movement, though sufficiently accurate for time and general amplitude, cannot be used for accurate quantitative analysis. The latency of movement was tested separately and found to vary from 0·004 sec. with short tubing (head, trunk and upper limb) to 0·01 sec. with longer tubing (lower limb). In records as rapid as those in figs. 25–29 there was no measurable difference between air transmission and direct beam from muscle. Time was recorded by a rotary Rayleigh wheel driven by an electrically operated tuning fork. When action currents have been recorded a Matthews oscillograph system has been used, with leads from the muscle by needle electrodes.

PROTOCOLS

Case I. Regular series of predominantly left-sided convulsions at brief intervals. Previous history of sensory and inhibitory attacks. No loss of consciousness. Pain in affected limbs. Spastic left-sided weakness due to right parietal astrocytoma.

M.B., age 54 years, female, was admitted under the care of Dr. Gordon Holmes in November, 1932. There was no pertinent family history or previous illness. In September, 1922, there occurred the first of a long series of brief attacks at intervals from several days to several weeks. In each of these there occurred a sensation of something rushing over her head, lasting several seconds, during which she could neither speak nor move. There was no loss of consciousness, and she would be observed only to stand still with fixed expression. At times the patient would turn round during an attack, and on some occasions the left arm and leg would become somewhat stiff, at times only the left leg. In other attacks there was a pricking sensation of the left side of the neck and tongue. The frequency of these attacks was much reduced by luminal, though they continued at long intervals for the next three years.

In one night in May, 1925, there occurred four generalized convulsions with loss of consciousness and preceded for a short interval by a severe pain in the left leg ' as

though someone were trying to pull it out of its socket.' She was not incontinent nor did she bite her tongue. She was admitted to another hospital and a right parietal decompression performed, after which the left arm and leg were completely paralysed but recovered in four weeks. Attacks of varied pattern continued. For a brief interval she had warnings of the 'wave passing over her head,' but from that time onwards she had no aura. There were attacks of tonic and then clonic spasm in the left arm and leg, attacks in which she was unable to use the left arm for a few seconds, and also attacks in which the left leg would feel quite numb for perhaps a half-minute. In none was there loss of consciousness, and the attacks recurred at intervals of a few hours to a few weeks, the intervals being longest when she was taking luminal.

On November 15, 1932, there developed a numbness of the left arm and leg, which lasted continuously for five days, and the patient then commenced to have very frequent attacks of spasm of flexion, lasting 15 seconds to one minute, at the left hip and knee, accompanied by severe pain in the left thigh. As the attacks continued progressive loss of voluntary power in the left lower limb occurred. There was no involvement of the right side. At the onset of later attacks the left arm was drawn up, but she was still able to move the fingers.

Attacks observed on November 22, 1932, presented the following features: If the patient were talking speech ceased suddenly and her face stiffened. There was a variable degree of stiffening of both arms and the right leg; the left lower limb became tonically flexed at the hip and knee with fine jerking movements at the ankle. After some 10 to 12 seconds the jerking increased in amplitude and clonic movements at the hip and knee commenced and lasted about 15 seconds. In some attacks the left arm adducted strongly. The patient was able to move the right arm to order during the attacks and stated that she was fully conscious and suffering pain in the dorsum of the left foot and in the left thigh.

There was slight bilateral papilloedema and slight weakness of voluntary movement of the left side of the face. All movements in the left upper limb, especially those at shoulder and elbow, were weak, and there was spasticity in the adductors of the shoulder and flexors of wrist and elbow. There was no power of voluntary movement in the left lower limb, the extensors of which were extremely spastic. The tendon reflexes of the left limbs were exaggerated and the left plantar response was extensor. There was no defect of sensation. The decompression was under no increase in tension. The cerebrospinal fluid showed no abnormality.

She was treated by luminal gr. 2 three times daily. On November 20 she had 60 attacks, the following day 30, and on the day of examination 50 attacks. Her temperature was irregular, rising from 97·4° to 99·6° F.

Subsequently, on November 25, the previous decompression opening was explored by the late Sir Percy Sargent and the cerebral cortex of the parietal lobe was found to be replaced by gelatinous tissue which extended to the median longitudinal fissure. A mass about 2 inches in diameter was removed and histological examination proved it to be an astrocytoma. She made a good recovery from operation and was discharged from hospital with a spastic weakness of the left arm and leg.

Analysis of attacks recorded on November 22, 1932.—In this patient besides a manometer to record the pressure of the cerebrospinal fluid through the lumbar puncture needle it was arranged that the movements of the upper abdomen and of the left great toe were recorded, together with the fluctuations in pressure at the decompression opening on the right side of the head. We had the kind assistance of Dr. Gilbert Hall in recording the attacks. Seven fits were recorded in their entirety, and the characteristic records shown

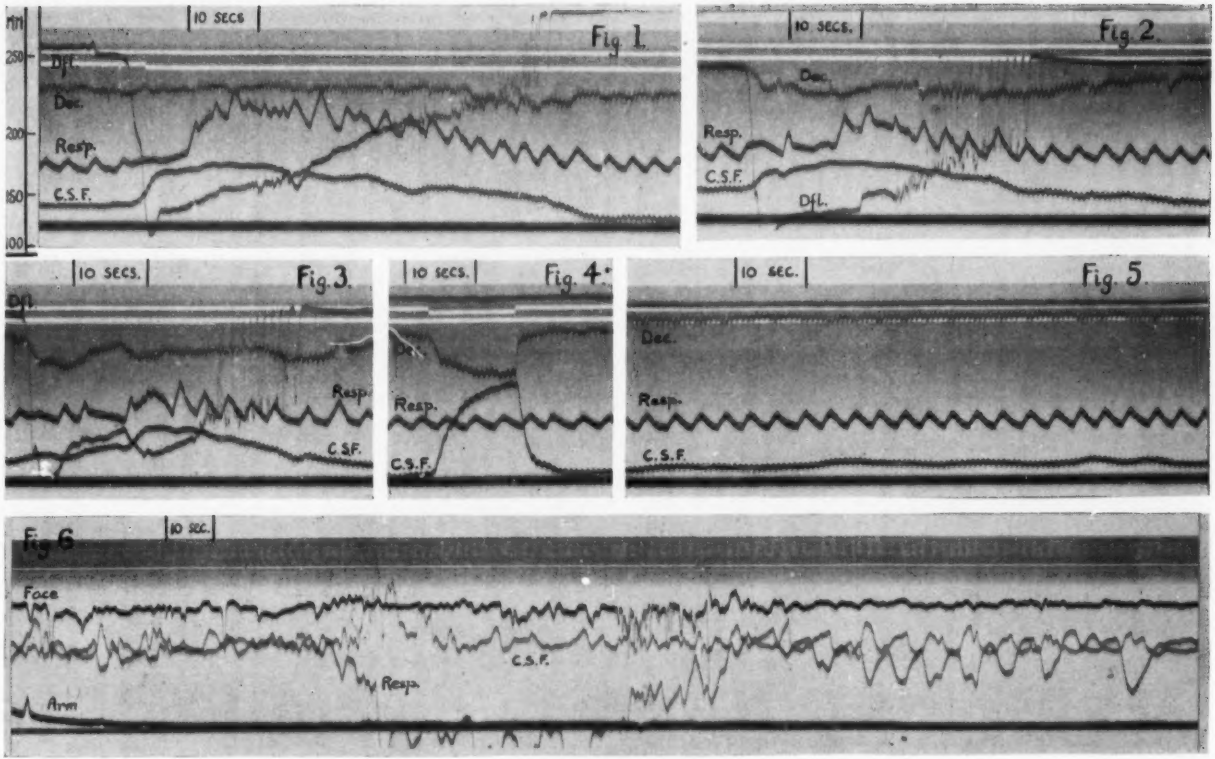

mm
250
200
150
100
10 secs
Fig. 1.
Dfl.
Dec.
Resp.
C.S.F.
10 secs.
Fig. 2.
Dec.
Resp.
C.S.F.
Dfl.
10 secs.
Fig. 3.
Dfl.
Resp.
C.S.F.
10 secs.
Fig. 4.
Dec.
Resp.
C.S.F.
10 sec.
Fig. 5.
Dec.
Resp.
C.S.F.
Fig. 6
10 sec.
Face
Resp.
C.S.F.
Arm

PLATE I.

in figs. 1, 2 and 3 show the course of the attacks. As will be seen in all these figures the onset of the attack was an abrupt dorsiflexion of the great toe, which was in all instances succeeded by a momentary cessation of respiration shown by the abdominal recorder. The cerebrospinal fluid pressure commenced to rise, usually as early as four-fifths of a second after the onset of dorsiflexion of the great toe (figs. 1 and 2), although in isolated instances, such

---

DESCRIPTION OF PLATE I

Plates I–VII. From records of epileptic attacks made photographically by movements of beams of light on a moving strip of bromide paper. All records read from left to right. Time is recorded electrically in fine vertical strokes at the top of the record at one-fifth second intervals. As the closeness of the vertical strokes in slow records tends to make them illegible after reduction, intervals of 10 seconds have been superimposed for the convenience of the reader. In Plates VI and VII the speed of the record renders this unnecessary. The tracing of the manometer recording cerebrospinal fluid pressure is marked C.S.F. in all records. In figs. 8, 11, 13, 14 and 15 the cerebrospinal fluid pressure is recorded both by delicate and coarse manometers the tracings of which are marked C.S.F. 1 and C.S.F. 2 respectively. The scale of tension at the commencement of each record refers to the calibration of the cerebrospinal fluid pressure (in mm. of water) as registered by the delicate manometer. The tracing which records chest movement is marked Resp. (or in some cases where abdominal movement was recorded, Abd.). The deflection is downwards for inspiration in all cases. In each case a record is made of some muscular movement prominent in the attack and the lettering of these records is described in the legend of each figure.

Plate I. Fig. 1.—Record of epileptic attack in Case I, 12 minutes after the last preceding attack. The uppermost moving line Dfl. registers movements of the left great toe and makes downward excursion for dorsiflexion and small upward excursion for plantar flexion. The tracing Dec. records the pressure within a balloon bound over the decompression opening in the skull, and shows an excursion with each pulse beat, and was also affected by movements of the face and neck. The pressure within the balloon at the commencement of the record was 12 cm. of water. The small movement of the foot three seconds before the commencement of a fit was probably voluntary.

Fig. 2.—Tracings and C.S.F. tension scale as in fig. 1, here showing a fit of shorter duration, 10 minutes after that recorded in fig. 1 and two and a half minutes after an intervening fit. The higher initial cerebrospinal fluid pressure was consequent upon some apnœa occurring after the hyperventilation of the intervening fit.

Fig. 3.—Tracing and C.S.F. tension scale as in fig. 1. Two minutes 40 seconds after the attack featured in fig. 2, showing delayed rise in cerebrospinal fluid pressure.

Fig. 4.—Tracings and C.S.F. tension scale as in fig. 1. To show the effect of compression of the left jugular vein in the interval signalled by the duration of the fall in the upper white line.

Fig. 5.—Tracings and C.S.F. tension scale as in fig. 1. To show irregular fluctuations in the cerebrospinal fluid pressure during a period of drowsiness some 10 minutes after the attack recorded in fig. 3.

Fig. 6.—Showing myoclonic twitching followed by major attack in Case II. The uppermost darker tracing (Face) shows a downward deflection for contraction of the left corner of the mouth. The figure commences with the occurrence of the first movement after a long period of quietude. Following the period shown in this figure the twitching disappeared after a few seconds and hyperpnœa occurred before the patient again became quiet. In the centre of the figure contraction of the trunk muscles in the tonic stage of the convulsion causes prolonged displacement of the chest recorder (Resp.). C.S.F. pressure as in scale in fig. 7.

as fig. 3, the rise was much longer delayed. This rise of pressure amounted at times to as much as 65 mm. of cerebrospinal fluid, as in fig. 1. After a first rapid rise it usually maintained an almost steady plateau, commencing to subside only when abrupt irregularity in the toe-recorder showed the first evidence of the clonic stage. Whether regular beats of clonus continued from this time onwards, as in fig. 2, or became established only very late in the attack, as in fig. 1, the reestablishment of original cerebrospinal fluid pressure was gradual and progressive. As will be seen in all three records the later fall of fluid pressure is related more directly to the reestablishment of regular respiration, as shown by the abdominal recorder. Where the abdominal movement at the onset of the attack is slight, as in fig. 3, the cerebrospinal fluid pressure may rise rapidly, late in the tonic stage, when the abdominal and thoracic muscles eventually participate in the attack.

The attempted record of the pressure at the decompression opening was found to be partly independent of the cerebrospinal fluid pressure change, as is seen in the figures shown, and inspection at the time revealed that, however applied, the fixation of the balloon at the decompression orifice was affected by the powerful movement of the face and neck musculature, which in most attacks commenced to occur within from three-fifths to one second after the first movement of the great toe. The absence of any real independence in cerebrospinal fluid and intracranial pressure is shown by light jugular compression when particular care was exercised not to disturb the fixation of the balloon over the decompression (fig. 4).

It will be noted that the resting cerebrospinal fluid pressure varied from 135 to 150 mm. of fluid owing to the continual fluctuations which were observed during the intervals between fits. These were particularly marked when the patient was drowsy, as in fig. 5, but in this patient were not observed to be greater than 20 mm. in range. They were not accompanied by any parallel change in respiration or abdominal posture. In one attack the pressure following the fit remained higher (110 mm.) than it was before the attack (100 mm.). In the other attacks, as in figs. 1, 2 and 3, the pressure was lower for an interval of some 15 to 30 seconds following the attack. There was no change preceding the attack in any of the records.

Case II. Generalized convulsions from early age. Three periods of status epilepticus at long intervals. Epileptic myoclonus, with 'reflex' provocation. Major attacks with predominantly left-sided convulsion. Repeated development and recovery of postepileptic hemiplegia.

G.T., age 12 years, female, admitted under the care of Dr. Gordon Holmes, in July, 1931, May, 1932, and January, 1933. No family history of epilepsy. Generalized convulsions commenced at the age of four years, and continued at the rate of two to four a week. The attacks were almost always nocturnal, usually with biting of the tongue and rarely with incontinence. Luminal administered regularly controlled the attacks, but the parents refused to continue its use. In July, 1931, generalized

convulsions, with loss of consciousness, incontinence and biting of the tongue, commenced to occur at hourly intervals. There was sufficient recovery in the intervals to talk, but after two weeks the left upper and lower limbs were noticed to be weak between the attacks. A week later, when she was admitted to hospital, the attacks were still occurring at the rate of about one an hour, each followed by a drowsy stage which lightened only for a brief interval before the onset of the next attack. The left limbs were weak, particularly in the grasp of the hand and in dorsiflexion of the foot, and both were slightly hypertonic, with increased tendon jerks. The abdominal reflexes were absent on the left side and the left plantar response was extensor.

In the attacks the head was turned to the left and the eyes upwards and to the right, the left arm and leg showed clonic movements, the right arm and leg rigidity. In some attacks twitching of the face preceded that of the left arm and leg. The evening temperature was usually elevated—on the thirteenth day, when the fits were lessening, reaching 102° F. Only once was the morning temperature high (99° F.). There was no correspondence between the number of attacks and the height of the temperature. She was treated by luminal and paraldehyde (fig. 30, status I). The intervals between attacks lengthened and the left hemiparesis gradually disappeared.

On discharge from hospital the patient remained free from attacks for one month and at the end of that period, at intervals of seven to 14 days, there commenced a series of momentary nocturnal attacks of twitching of the eyelids with generalized stiffness and trembling. Nocturnal enuresis was frequent. At the beginning of May, 1932, the frequency increased to two to three attacks every night and within a few days attacks became very frequent indeed, and generalized convulsions occurred. The smaller attacks now occurred in the daytime. In these, with the trembling there was a little cry, the lips would become blue, and she was unable to talk for a moment or two. The large attacks took the form of turning of the head to the right with flexion of the arms at the elbows, hands clenched, eyes closed, with some cyanosis of the lips. The head then turned to the left and the left arm extended. The attack subsided with some clonic jerking of the left arm and was followed by drowsiness. The total duration was some 30 seconds. In other attacks the head turned to the left with eyes to the right, the right upper limb was held extended, the left flexed. In the intervals between the attacks the left arm and leg were hypotonic, but there was at this time no defect in motor power, except for a left facial weakness. The tendon reflexes were increased in the left upper limb, but equal in the lower limbs. Abdominal reflexes were absent and plantar responses both flexor. Blood pressure 120/70.

Neither paraldehyde nor morphia controlled the attacks, which were temporarily reduced in number, but not controlled by luminal gr. 5 intravenously. The attacks diminished in severity but the total frequency was little altered. A left-sided hemiparesis developed, with marked left-sided hypotonia, and extensor plantar response. There was defective sensation of position and passive movement with astereognosis in the left hand. At this time records of the fits were made. The temperature rose only on two occasions, once in the evening to 99° and once in the morning to 99·8° F. (fourteenth and sixteenth days—fig. 30, status II). Treatment by bromides, chloral and luminal (up to gr. 4 a day by mouth) was continued and the number of attacks lessened and eventually ceased. Ventriculography showed no displacement or deformity of the ventricles.

Attacks continued at very rare intervals during the next six months, when again, in spite of continuous treatment by luminal, tonic convulsions of the left arm and leg commenced to become very frequent. On this occasion a left-sided hemiparesis, with flaccid arm and slight increase of tone in the lower limb, developed. There was loss of two-point discrimination and astereognosis in the left upper limb. The attacks were as before, except that both head and eyes were turned to the left. Respiration was profoundly affected. The left plantar response was observed on several occasions

to be extensor for a period of three minutes after an attack, the right remaining flexor throughout. The attacks gradually lessened in frequency and finally ceased (fig. 30, status III). The temperature showed only occasional elevation to 99·2° F. on the morning of the third day and to 99° F. on the evening of the twenty-sixth day. When the attacks were most numerous the temperature varied between 97° and 98·4° F.

On March 7, 1933, an exploration of the cerebral cortex by a right fronto-temporo-parietal osteoplastic flap was made by Mr. Julian Taylor. The subarachnoid lakes appeared to be unusually distended. There was no evidence of tumour. The patient made a rapid recovery, and has remained free from fits.

Analysis of attacks recorded on June 10 and 15, 1932.—The facial movements in this patient showed continued irregular brief twitchings occurring singly or in groups. At times twitching of the left arm and of the left side of the trunk also occurred. In records of these twitchings there appeared to be no direct relationship between the occasional myoclonic twitches in the face and those which were observed in the trunk and wrist as shown in figs. 6 and 8. At long intervals, usually when all twitching had gradually subsided in the preceding 10–20 minutes, abrupt and violent twitching would break out in the left side of the face, trunk and left arm concurrently. This would partly subside sometimes after five or 10 seconds, leaving for an interval more frequent twitching in the face. The cerebrospinal fluid pressure at these times showed only some small fluctuations corresponding to those of the chest. At other times, as in fig. 6, the initial abrupt movements in the face and trunk gave way to a more maintained tonic posture lasting up to a minute, during which time some isolated jerky movements of the face, arm and trunk occurred. For a variable period following this a rapid irregular clonic twitching of the face remained. With the initial outburst in such cases the cerebrospinal fluid pressure showed irregular jerks of increase, but fluctuations occurred corresponding in time to the larger trunk movements. In the intervening periods when the patient was drowsy, when myoclonic twitching was rarely observed or at times absent for periods of minutes, it was noticed that a sudden stimulus caused awakening accompanied by a rapid sequence of myoclonic twitches. In this way a myoclonic attack or even a tonic-clonic convulsion appeared to be elicited by stimulation. Such a convulsion is recorded in fig. 7. Here, a loud noise having proved ineffective, sudden cold was applied to the left calf. Immediately there resulted movement of the left arm and left leg and after two seconds rapid twitches of wide amplitude appeared in the left side of the face and the left limbs. These ceased after a further two seconds, when there was a powerful contraction of the left side of the trunk with cessation of respiration. After a cessation for some four seconds this tonic contraction was renewed in more powerful form and a tonic convulsion was established. The clonic stage which ensued passed into irregular myoclonic twitching lasting many minutes. The cerebrospinal fluid pressure undergoes small fluctuations which reflect only the more abrupt movements of the chest.

Case III. Two isolated outbursts of status epilepticus, with long intervening interval. Compulsion aura. Predominantly right-sided attacks with speech defect. Postepileptic paralysis. Asthmatic subject.

L. K., male, age 31 years, admitted to the National Hospital October, 1933, under the care of Dr. Hinds Howell. From his youth he had suffered from attacks of asthma, with occasional eruptions of giant urticaria. In October, 1932, without warning he had a generalized convulsion, with loss of consciousness, succeeded by stupor, which was interrupted 24 hours later by another convulsion. During the next nine days, of which he has no memory, he remained in a stuporose state. He states that in the subsequent few days he felt 'completely exhausted' and remembers having six to eight attacks in which his right arm jumped and the elbow flexed involuntarily. He then remained free of attacks until April, 1933, when he commenced to experience momentary 'sensations' in which he felt that he 'must do something but did not know what.' Two weeks later one of these sensations was followed by a generalized convulsion with loss of consciousness. Treatment with luminal was then commenced and he had no further attacks until a generalized convulsion followed two weeks after the discontinuing of such medication in June, 1933. He was admitted to hospital five days later and no abnormal physical signs were found. The cerebrospinal fluid was normal. Skin tests showed a moderate reaction to the meat group, in particular to beef. On discharge he remained well except for periodical asthmatic attacks, which were relieved by ephedrin. At the height of one such attack the occurrence of one of his 'sensations' was accompanied by subsidence of the asthmatic attack. On September 26, 1933, after momentary confusion of mind he lost consciousness for a period of two hours, after which he remembers twitching all over. His asthmatic trouble, previously occurring nightly, was lessened for 48 hours. Four days later the patient had an attack in which he felt impelled to say something but was unable to speak, following which his right arm was raised. Similar attacks followed, and in the intervals, which became progressively shorter, he felt exhausted and had difficulty in expressing himself. In this state, two days later, on October 2, when the attacks were recurring every 20 minutes, he was admitted to hospital. The unpleasant sensation of necessity for speech, with the production of an unrecognisable sound which trailed off into 'a silence,' continued to precede each attack. He was able to understand what was said to him during an attack but could make no response of any kind. Observed attacks commenced with an irregular groaning, twitching of the upper lip, turning of the head to the right, and similar deviation of the eyes with occasional reassertions of forward gaze. His right arm became flexed at elbow and wrist. After two minutes the periodic groaning ceased, he would smile, and after a momentary clonic twitching of both corners of his mouth, he would turn his head forward and speak rationally. A series of such attacks was recorded.

Upon examination slight weakness of the lower right side of the face and of the right side of the palate and tongue were apparent. Motor power, tone and limb reflexes were normal, except that the right plantar response was less flexor than the left, and immediately after an attack was extensor for a few minutes. The cerebrospinal fluid contained 20 cells per c.mm., total protein 0·030 per cent.; globulin tests were negative and the Lange normal. Ventricular fluid contained 19 cells per c.mm. and protein 0·03 per cent. Ventriculography failed to reveal any abnormality of the cerebral ventricles.

The day after his admission some attacks involved the face alone, and while the right side was drawn up in spasm the left eye would close intermittently and the mouth would be drawn first to one side and then to the other. In some attacks the right arm was involved in clonic convulsion, in others in tonic flexion with tonic extension of the

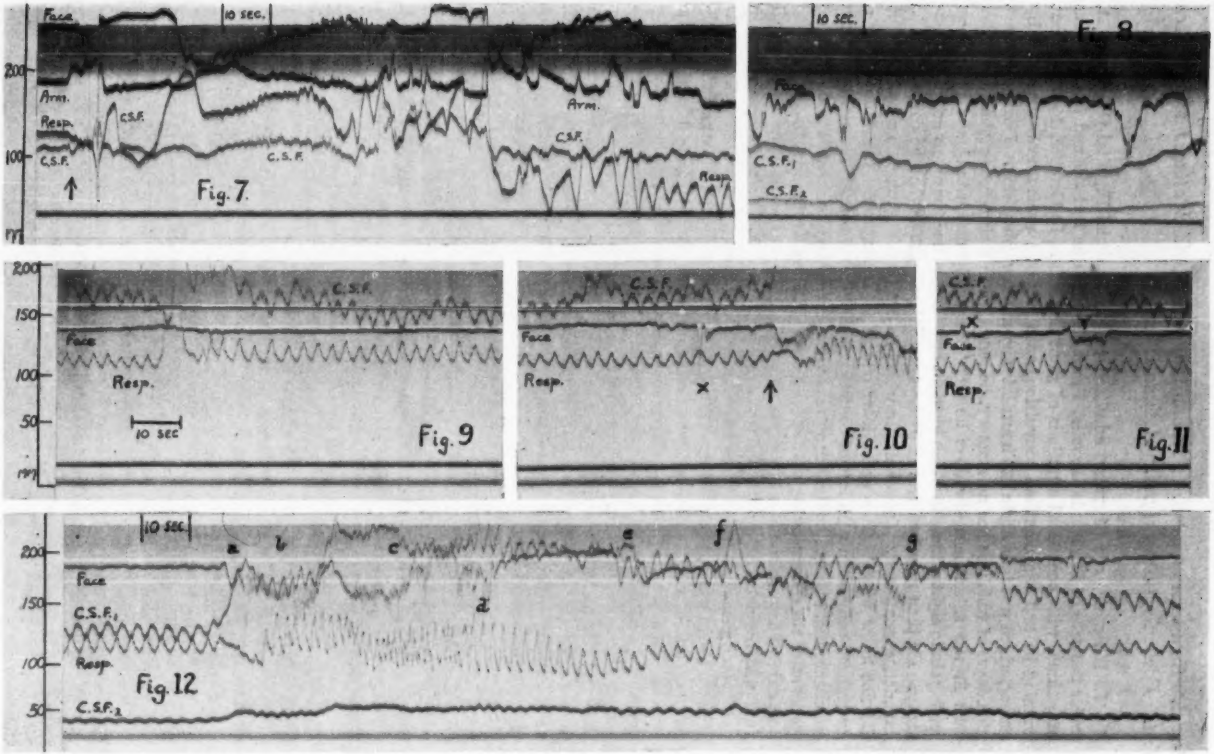

Face
10 sec.
200
Arm.
Resp.
C.S.F.
100
Fig. 7.
Fig. 8
C.S.F.1
C.S.F.2
150
50
Fig. 9
Fig. 10
Fig. 11
X
Y
a
b
c
d
e
f
g
Fig. 12

PLATE II.

right leg. After some attacks he was completely aphasic for 10 minutes. Examination now showed increased weakness of the right side of the face, palate, tongue and right hand. The right arm was hypotonic and both lower limbs showed a slight increase in extensor tone. The tendon reflexes were equal and brisk in the lower limbs, with patellar clonus; the plantar responses were extensor during the attacks but plantar flexion replaced this reaction in the intervals. On the day of admission he had 33 attacks and three drams of paraldehyde were administered per rectum. The following day he had 26 attacks and was treated by two hypodermic injections of morphia (gr. $\frac{1}{6}$ and gr. $\frac{1}{4}$), 4 drams of paraldehyde per rectum, and luminal gr. $1\frac{1}{2}$ three times daily. The last was alone continued thereafter. On October 4 he had nine attacks, on October 5, five, and then was free from attacks during the rest of his stay in hospital. There was no elevation of temperature with the attacks.

Analysis of attacks recorded on October 2, 1933.—In this patient the use of a wide-bore lumbar puncture needle and a sensitive manometer enabled us to obtain a more than usually clear record of the pulse and respiratory waves. The close correspondence between the movements of the chest and the cerebrospinal fluid pressure is evident in fig. 9, where for a brief interval the patient is instructed to cough. Even speech, to the extent of the enunciation of one word, also causes a brief rise of pressure (fig. 11). The initial fall, and after a brief interval the sudden rise in cerebrospinal fluid pressure, are distinctly seen, also the subsequent slight fall of pressure for a period of some 30 seconds due to the increased movement of the chest with increased ventila-

PLATE II.

DESCRIPTION OF PLATE II

Plate II. Fig. 7 (Case II).—As in fig. 6, with the addition of a tracing (Arm) recording occurrence of flexion of the left wrist as an upward excursion. The patient having previously been asleep, cold is applied to the left calf at the moment indicated by the arrow directed upwards. A powerful myoclonic jerk is followed after a few seconds by the commencement of an attack in which tonic movement of the face with expiration is followed by a long period of myoclonus.

Fig. 8.—Tension scale for C.S.F. 1 as in fig. 7. The upper record shows by downward deflections a series of myoclonic contractions of the left side of the face. Respiration is not recorded.

Fig. 9 (Case III).—To show the effect of a cough in the duration of fall of the upper white line.

Fig. 10 (Case III).—Time, C.S.F. pressure, and respiration as in fig. 9. To show the earliest change in C.S.F. pressure as recorded by an extremely sensitive manometer. A balloon over the right corner of the mouth causes a manometer tracing (Face) to move downwards when contraction occurs. At the point marked X the patient made some small voluntary facial movement while at the point marked (↑) a convulsion commences.

Fig. 11.—C.S.F. tension, etc., as figs. 9 and 10. To show effect of two brief periods of speech marked X and Y.

Fig. 12.—Complete record of a fit in Case III. Time, C.S.F. tension and other records as in figs. 9, 10 and 11, with the addition of a tracing of a coarse manometer C.S.F. 2. Some momentary displacements of the upper white line are made by an electrical signal by the observer to indicate at *a* slight turning of the head towards the affected side, at *b* the first groaning sound, and at *c* more powerful turning of the head to the affected side; at *d* the patient nodded his head and said 'I'm all right.' From *e* to *g* the patient talked and at *f* coughed to order.

tion immediately following the apnœa. Jugular compression was followed by a rapid rise of cerebrospinal fluid pressure within two seconds. This manometer showed well the earliest changes during a fit (fig. 10), and a fully recorded fit with both coarse and delicate manometers is shown in fig. 12. The pressure is seen to commence to rise apparently as the first event in the attack, although close inspection of the record reveals that the accompanying respiration is more brief than its predecessors. The tonic stage of the attack lasts only five seconds in the face, and the steep rise of cerebrospinal fluid pressure during this interval thereafter ceases, falling very slightly during subsequent clonus and rising sharply again 15 seconds later where the facial movements again become tonic for a brief interval. The period of apnœa here outlasts the initial rise of pressure and at the end of the attack the pressure falls only very slowly, being delayed for some 20 seconds by a renewed outburst of clonic twitching of the face. The original level was reached only after four minutes from the termination of the attack. The recorded signals during this fit indicate that vocalisation occurred with or slightly after the first movement of the face and that the secondary rise of cerebrospinal fluid pressure begins seven seconds after the commencement of turning of the head. It will be noted that the regular rapid movements of the chest show no change at this time. All these features were confirmed in the record of another fit.

Case IV. Onset of epilepsy three months after the evacuation of a frontal abscess. Three periods of status epilepticus. Left-sided attacks, with post-epileptic paralysis and sensory disturbance. Coincidence with reflex stimulation.

L. S., age 27 years, female, admitted under the care of Dr. James Collier in March, 1930, January, 1931, and November, 1933. In 1922 the patient had a septic appendix removed. In 1925 there began severe frontal headaches with failing vision. In January, 1926, she was admitted to St. George's Hospital where a right frontal cerebral abscess was drained. In March, 1926, she commenced to suffer from attacks of unconsciousness preceded by twitching of the left arm and leg, occurring at long intervals for four years, but gradually increasing in frequency. Two days before her admission to hospital on March 14, 1930, attacks involving the left arm and sometimes the left side of the face commenced to occur every few minutes. For one to two minutes after the cessation of such attacks the left arm was useless. The attacks were partly controlled by the administration of four-hourly doses of paraldehyde and ceased five days after admission. She had only one rise of temperature, to 99·8° F., during this time. The attacks then ceased completely, but bromide and luminal were continued. In January, 1931, attacks of shaking of the left arm lasting a few seconds and occurring every half-hour commenced. Three days later the attacks spread further; commencing with flexion of the left elbow and wrist and turning of the head to the left, the left side of the face and trunk were affected later in the attack. After the onset of the fit there was no movement of the head, which could be turned in the opposite direction to order. There was no deviation of the eyes. Consciousness was not lost. Again weakness of the left side of the face and the left arm, with diminished tone and slight weakness of the left arm, was observed in the intervals between the

attacks. There was complete astereognosis, loss of two-point discrimination and diminution of postural sensibility in the left hand.

The attacks were treated by paraldehyde and rapidly became fewer, and thereafter under luminal treatment she remained free from attacks until October, 1933, having had a pregnancy in this interval. A single nocturnal fit then occurred. A week later, after a day of infrequent attacks, the fits recurred every three to five minutes. Paraldehyde treatment controlled the attacks to some extent until the fifth day after their onset, when they commenced to occur every two to five minutes. The attacks continued during waking and sleeping hours for 14 days in spite of intravenous administration of luminal gr. 3 on three occasions and 2 drams of paraldehyde by rectum four-hourly. During this period there gradually developed a complete flaccid paralysis of the left side of the face, arm and leg. In the three weeks following cessation of the attacks the paralysis slowly improved, leaving slight weakness of the left side of the face, astereognosis in the left hand and loss of two-point discrimination in the left hand and left foot. The temperature reached 99° F. on one occasion only, after the cessation of the attacks.

At the time records were made of the attacks the sequence of events was as follows: For a second or two the patient appeared to be aware that an attack was imminent. Her head turned slightly to the left and she complained of a tingling sensation in the left hand. The left arm was abducted at the shoulder and partially pronated at the elbow, with extended wrist. The wrist then flexed and the fingers extended. The left leg was then abducted at the thigh and feet were inverted and extended. The right leg showed a similar movement of the foot only. The left side of the face was slightly contracted. When these attitudes had been maintained for 10 to 15 seconds clonic movements appeared in the left side of the face, in the left arm, abdominal muscles and slightly in the lower limb. Clonic movements were sometimes seen in the deep muscles of the right side of the neck. These movements appeared to occur in the same order as the tonic spasm and persisted longest, sometimes in the left hand, sometimes at the left corner of the mouth. The patient was conscious and able to speak during the attacks.

In the intervals between the attacks the left arm and leg were in a state of complete flaccid paralysis. There was no demonstrable weakness of movements of the upper face, but the left side of the lower face remained completely immobile upon attempted voluntary movement. For associated movements the whole left side of the face moved well but not so strongly as the right. On emotional expression the left side of the lower face moved earlier and more completely than the right. The palate was slightly weak on the left side and the tongue deviated to the left. There was no weakness of sternomastoids or trapezil. There was slight weakness of the abdominal muscles on the left side. The tendon jerks were increased in both arms, and both knee and ankle jerks were present and equal. The abdominal reflexes were absent on the left and present on the right. The plantar reflex was flexor on the right, absent on the left. In the upper and lower limbs there was complete loss of position sense, appreciation of passive movement, stereognosis and two-point discrimination, with loss of position sense and two-point discrimination in the left lower limb. The blood pressure was 170/110 mm. Hg. both during and between the attacks. The cerebrospinal fluid was reported to contain no cells, protein 0·050 per cent., Nonne-Apelt negative, Pandy positive. Lange and Wassermann reactions negative. The blood calcium was 10 mgm. per cent. in one period of attacks.

Analysis of attacks recorded on November 7, 1933.—In this patient attacks were occurring at brief intervals of surprising regularity. In consecutive attacks which were recorded the intervals from onset to onset were as follows:

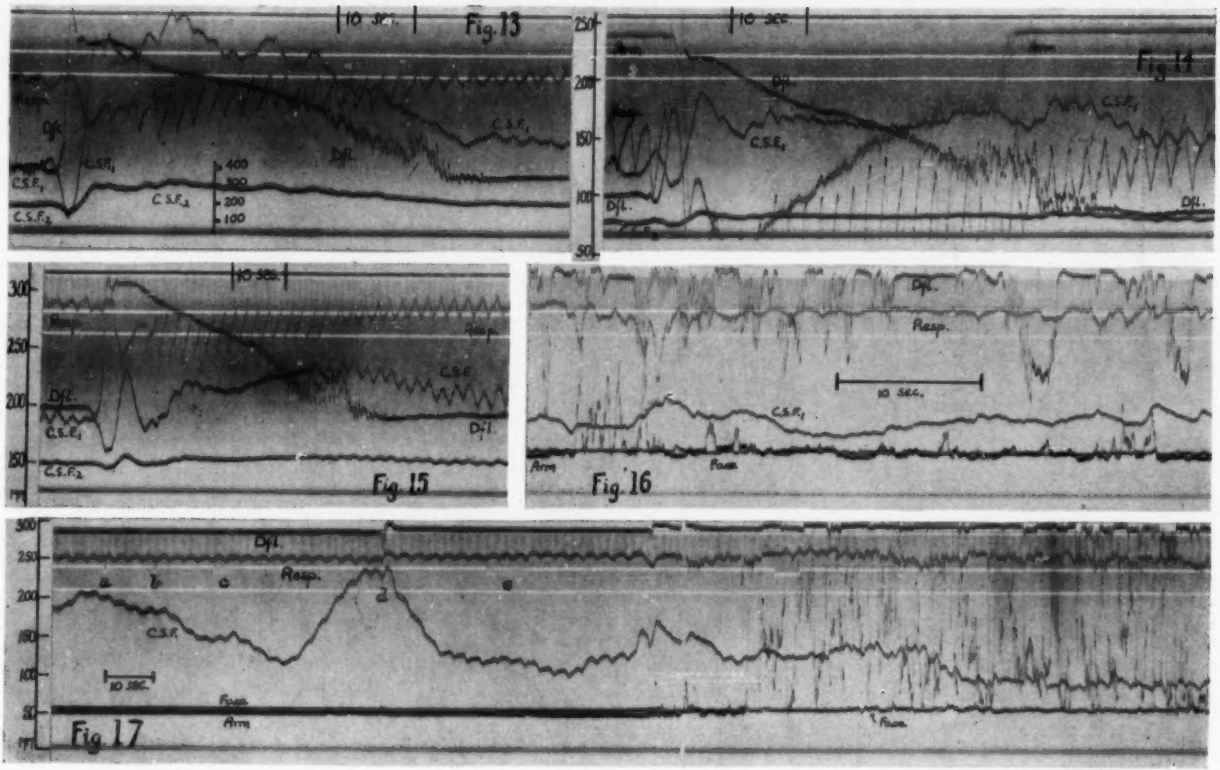

10 sec.
Fig. 13
Resp.
Dyl.
C.S.F.1
C.S.F.2
400
300
200
100
250
200
150
100
50
mm
10 sec.
Fig. 14
Dyl.
C.S.F.1
C.S.F.2
Dyl.
Dyl.
300
250
200
150
mm
10 sec.
Resp.
C.S.F.
Dyl.
C.S.F.1
C.S.F.2
Dyl.
Fig. 15
Dyl.
Resp.
10 sec.
C.S.F.1
Arm
Resp.
Fig. 16
300
250
200
150
100
50
mm
Dyl.
a
b
c
Resp.
d
C.S.F.
10 sec.
Resp.
Arm
Resp.
Fig. 17

PLATE III.

3 min., 3 min. 8 sec., 3 min. 8 sec., 3 min. 30 sec., 3 min. 54 sec., 3 min. 36 sec., 3 min. 36 sec.; after two attacks in which the interval was not recorded, a further consecutive series showed the following intervals: 4 min. 3 sec., 3 min. 22 sec., 3 min. 25 sec., 3 min. 37 sec., 3 min. 32 sec., 3 min. 28 sec., 3 min. 12 sec., 3 min. 28 sec., 3 min. 48 sec., 3 min. 32 sec. The slight variation was not accounted for by variation in the duration of each attack, which showed independent slight fluctuation.

In these attacks, examples of which are shown in figs. 13, 14 and 15, there was constantly an initial fall in cerebrospinal fluid pressure followed by a rapid and high rise, which thereafter subsided gradually during the clonic stage of the attack. The initial fall in pressure was usually, but not always, preceded by a slight plantar flexion of the foot. It was regularly preceded by a somewhat deeper inspiration than before, and the succeeding sharp rise was contemporaneous with a very deep expiration, which heralded the onset of the tonic stage of chest movement. This relationship is clearly shown in fig. 14. In all attacks the tonic chest movement subsided more rapidly than the tonic stage in the limbs. Where the movement of the arm was recorded, as in fig. 14, it was found that the clonic stage ceased earlier in this member than in the lower limb. Particular care was taken to protect the decompression opening in the skull from any adventitious pressure. The cerebrospinal fluid pressure returned to its previous level only some 30 to 60 seconds after the termination of the attack. It will be noticed that the delay in the final

DESCRIPTION OF PLATE III

Plate III. Fig. 13 (Case IV).—The scale of tension recorded by C.S.F. 1 is shown at the commencement of fig. 15. The dark tracing Dfl. moves upward with dorsiflexion of the left foot.

Fig. 14.—As fig. 13, with the addition of a manometer (Arm) which by its downward deflection shows the occurrence of clasping of the left hand. Though by observation the attack appeared to commence at the same moment in foot, hand and face, this record reveals some delay in the movement of the hand.

Fig. 15 (Case IV).—As fig. 13. To show a more brief attack, with less initial rise of cerebrospinal fluid pressure.

Fig. 16 (Case V).—Tension of cerebrospinal fluid as in fig. 17. To show by more rapid recording the relationship between the individual clonic movement in face, arm, thorax and foot. Time intervals in one-fifth second are seen as vertical white strokes at the top of the record. The uppermost tracing Dfl. shows downward movement for dorsiflexion of the left foot. Below it (Resp.) movements of the thorax are recorded with downward displacement for inspiration. Below the C.S.F. pressure tracing (C.S.F.) two superimposed lines show movements of the face and arm. The darker (Arm) shows clasping of the hand; the lighter (Face), which makes only small deflections, records contraction of the left corner of the mouth.

Fig. 17 (Case V).—Shows the commencement of a generalized myoclonic convulsion following stimulation of the patient during sleep. The figure begins with high C.S.F. pressure during sleep. At the signal recorded in the upper white line at *a* the dorsal aspect of the left forearm was pricked once, at *b* once, at *c* 10 times in succession, again at *d*, and 12 times at *e*.

fall of pressure is often related to a similar delay in the return of the chest wall to the depth of full expiration.

The effect of various stimuli which appeared to be provocative of attacks was recorded. Friction of the left hand and stimulation of the sole of the left foot were tried on many occasions, on most of which they were without effect, and when they were succeeded by an attack the time of the attack was within the limits of natural variation of the intervals above noted.

Case V. Congenital syphilis. Rapid development of dementia with involuntary twitching, the latter improved by treatment. Recurrence six months later with right-sided epileptic seizures and rapid onset of right hemiplegia. Subsequent onset of left-sided myoclonic convulsions.

W. S., age 19 years, male, admitted under the care of Dr. Hinds Howell in February, 1934. He was born at full time of apparently healthy parents who had previously had three healthy children and one miscarriage. At the age of five years it was discovered that the right eye was blind and the vision of the left eye impaired. When eight years old he attended a school for the blind and made satisfactory progress. At the beginning of 1933 he was noticed to make purposeless movements of his tongue, and he had difficulty in speaking, especially when excited. In May, 1933, unsteadiness in walking and purposeless movements of his hands and legs were noticed. In July, 1933, he was admitted to hospital. His speech was almost unintelligible owing to dysarthria and he showed an advanced degree of mental impairment. In the right eye there was no perception of light : the visual acuity of the left eye was 6/36 corrected. The right eye was small with a band of corneal degeneration, probably congenital. The left eye showed some synechiæ from old iritis and a widespread syphilitic choroidoretinitis. There was constant nystagmus of moderate rate and equal components, both at rest and on lateral deviation of the eyes. The left pupil reacted to direct light. Convergence was poor. The tendon reflexes were exaggerated and he showed irregular choreiform movements of the limbs and constant grimacing. The cerebrospinal fluid showed 42 cells, total protein 0·060 per cent., globulin positive, Lange 4544332100. Wassermann reaction full positive in cerebrospinal fluid and blood. After a course of intravenous therapy speech was a little clearer and the involuntary movements were less pronounced.

In November, 1933, a left hemiplegia occurred suddenly, with complete recovery during the ensuing night. He then remained well until the end of January, 1934, when for a period of 24 hours he developed extreme restlessness of both upper limbs and trembling of the lower limbs. Three days later he again had difficulty in moving his legs and after two further days, on February 4, at frequent intervals, for 24 hours, the right arm and leg commenced a series of spasms each lasting about 10 minutes, with the gradual development of a complete right-sided hemiplegia followed by cessation of twitching. At that time, on the morning of admission, the left corner of the mouth and the left hand commenced to twitch and there was occasional vesical incontinence. He was admitted to hospital in a stuporous condition. Attacks of twitching were observed in the left corner of the mouth and left eyelids ; more rarely there was twitching of the jaw. The left hand was rarely quiescent, displaying purposive movements, and in addition irregular myoclonic movements of the fingers, or of one finger alone, and occasionally of the wrist, elbow and shoulder. All movements were much increased by interference, which promoted a directed resistance, and ceased during periods of quietude. Occasionally the left thigh flexed or abducted and the

great toe dorsiflexed. The left upper and lower limbs were of normal tone and no defect of muscular power was demonstrable. Reflex grasping could be elicited by stimulation of the palm of the left hand. The right side was completely paralysed and immobile, with increased flexor tone in the upper limb and extensor spasticity in the lower limb. The tendon reflexes were present and equal in the upper limbs, brisk and equal in the lower limbs, the abdominal reflexes were absent on the right side, and both plantar responses were extensor. Cooperation for sensory examination was not possible, but pin-prick evoked more general reaction from the left side than from the right. The cerebrospinal fluid was reported to contain 23 cells per c.mm., total protein 0·037 per cent., globulin weakly positive, Lange 1223442210. Wassermann reaction full positive in cerebrospinal fluid and blood.

On February 6 four drams of paraldehyde per rectum reduced the involuntary movements to occasional clonic twitches of the left side of the face and the left hand, which still occurred in greater number, together with resistive movements of the left arm when the patient was disturbed. His temperature varied between 100·2° and 103° F., his pulse between 108 and 138. Minor attacks continued in spite of three further administrations of four drams of paraldehyde, until his death two days after admission. A postmortem examination disclosed considerable dilation of all cerebral ventricles due to granular ependymitis, with chronic arachnoiditis, and accompanied by considerable cortical degeneration (dementia paralytica).

Analysis of attacks recorded on February 5, 1934.—In this patient the cerebrospinal fluid pressure was found to be undergoing wide fluctuations during the intervals of sleep or stupor between the attacks (figs. 18–21). There was no accompanying fluctuation of respiration. The maximum pressure was 300 mm., with falls to 160 mm., lasting 40 seconds, as at *a* in fig. 18, with considerable fluctuation in extent and duration. Before the onset of an attack the average pressure would fall gradually and slowly to about 150 mm. Rapid irregular movements of the face, of the foot and of the chest would commence concurrently and increase in irregularity, the pressure at first showing only fine rapid fluctuations corresponding to the movements of the chest. After an interval of some 30 to 40 seconds the movements of the chest would become more violent and the pressure rise slightly. About this time rapid irregular clonic movement commonly commenced in the upper limb. The subsidence of the attack usually began with subsidence of the facial movements, but isolated irregular twitching of face, arm, trunk and leg continued to occur for many seconds, with sometimes an irregular fresh outburst in the arm or trunk, as in figs. 19 and 20. With the subsidence of twitching the pressure at first fell to the region of 100 mm. of water for an interval and rose in a series of increasing fluctuations to reach its original high level. The patient was at this time noted to be returning to his original sleeping state from which the attack had awakened him.

Rapidly moving records of the twitching during an attack showed the complete independence of the myoclonic twitching in the face, arm, trunk and leg (fig. 16). The series of events recorded in figs. 18–21 was repeated in two other complete records of attacks, differing only in the relative violence and duration of movement. Occasionally a few myoclonic twitches of the arm

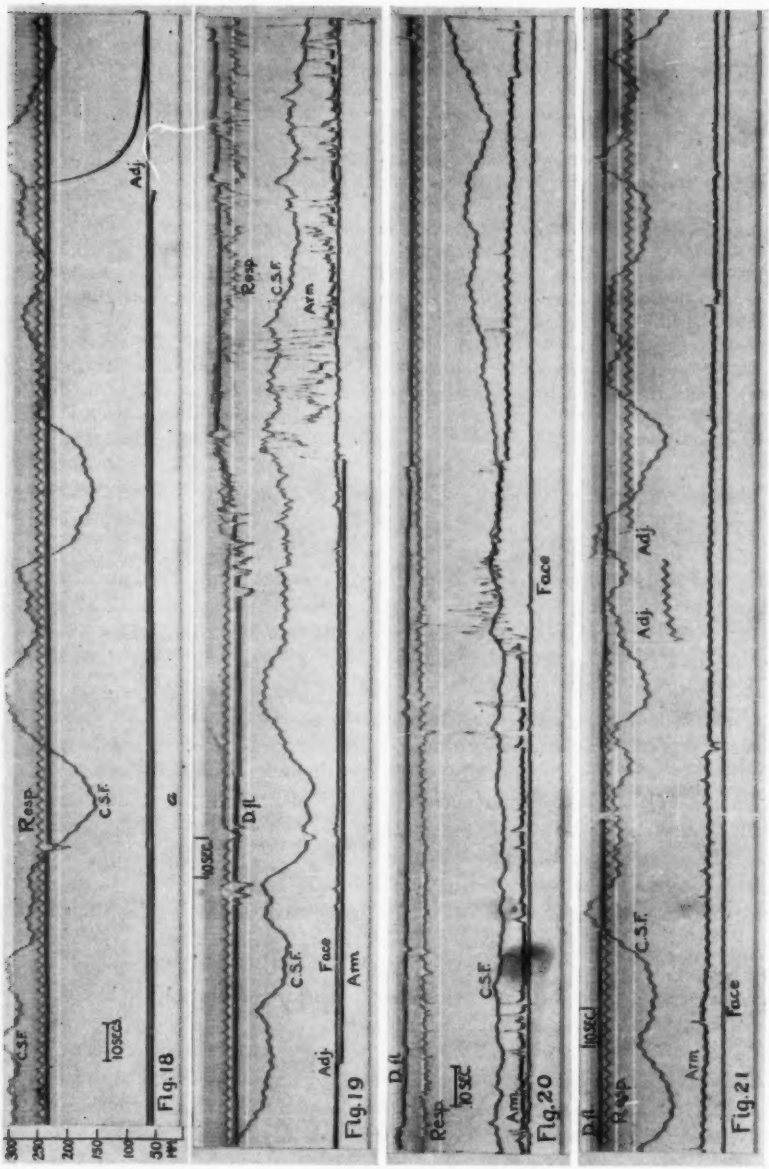

C.S.F.
Resp.
C.S.F.
10sec.
Adj.
a
Fig.18
10sec.
Resp.
C.S.F.
Dft.
C.S.F.
Arm.
Adj.
Face
Arm
Fig.19
Dft.
Resp.
10sec.
C.S.F.
Face
Arm.
Fig.20
Dft.
10sec.
Resp.
C.S.F.
Adj.
Adj.
Arm.
Face
Fig.21

PLATE IV.

appeared spontaneously in the trough of some of the larger fluctuations in the drowsy state. Such a ' minimal fit ' is shown in fig. 22. During the stuporose state stimulation, for example, by pricking the forearm, resulted in the gradual awakening of the patient with fall of the cerebrospinal fluid pressure in a series of wide fluctuations. When the pressure was low myoclonic twitching would commence abruptly, as in the spontaneous attack (fig. 17).

A loud clap of the hand during the drowsy state did not provoke myoclonus, although almost any stimulus increased the myoclonus when present. Inhalation of amyl nitrite during the height of an attack did not modify the cerebrospinal fluid pressure and affected the movements only in that they were slightly more violent for a brief interval. The rise of cerebrospinal fluid pressure during a fit appeared to be directly related only to movements of the chest and upper limb. Removal of 6 c.c. of cerebrospinal fluid did not modify the course of the attack. At the moment of removal the pressure was 240 mm., the previous fluctuations being small and reaching to 200 mm., the pressure after removal was 140 mm., and rose after 20 seconds to 200 mm., falling to 180 mm. After removal of another 6 c.c. the pressure remained steady for 30 seconds at 100 mm.

Case VI. Generalized convulsions at long intervals for seven years. For three years more frequent left-sided attacks, commencing in the left lower limb, without loss of consciousness. Continuous twitching of left foot for three months, with occasional spread to whole left side. Slight spastic weakness left lower limb. No organic cause found on cerebral exploration.

E. S., age 43 years, female, admitted under the care of Dr. Adie in March, 1934. No family history of epilepsy. Previous health good. Eleven years earlier, three months after the birth of her last child, she had a mild febrile illness during which there was an attack of unconsciousness with incontinence of urine. Twelve months later she again had an attack of unconsciousness with incontinence of urine. She was unable to say whether she had a generalized convulsion. From that time until three years before admission at long intervals similar attacks occurred. In one of these the tongue was bitten. In the last three years the attacks were more frequent, and more especially related to menstruation. In these without warning the toes of her left foot would become plantar-flexed, the left lower limb would be drawn up in a jerky manner, the left side of the abdomen and chest would become involved, then the upper extremity

DESCRIPTION OF PLATE IV

Plate IV.—Figs. 18, 19, 20 and 21 (Case V) form one continuous record of the transition of events from sound sleep at the commencement of fig. 18, to drowsiness at the commencement of fig. 19, a prolonged myoclonic convulsion in the latter half of fig. 19 and the first part of fig. 20, and finally to sleep in the latter half of fig. 21. The cerebrospinal fluid tension (C.S.F.) for all figures is shown at the commencement of fig. 18. Movements of the foot, face and arm are shown by D.fl., Face and Arm as in fig. 16. At Adj. at the end of fig. 18 and the commencement of fig. 19 the tension of the arm balloon was adjusted but without disturbance of the patient, as was also the respiratory recorder at Adj. in fig. 21.

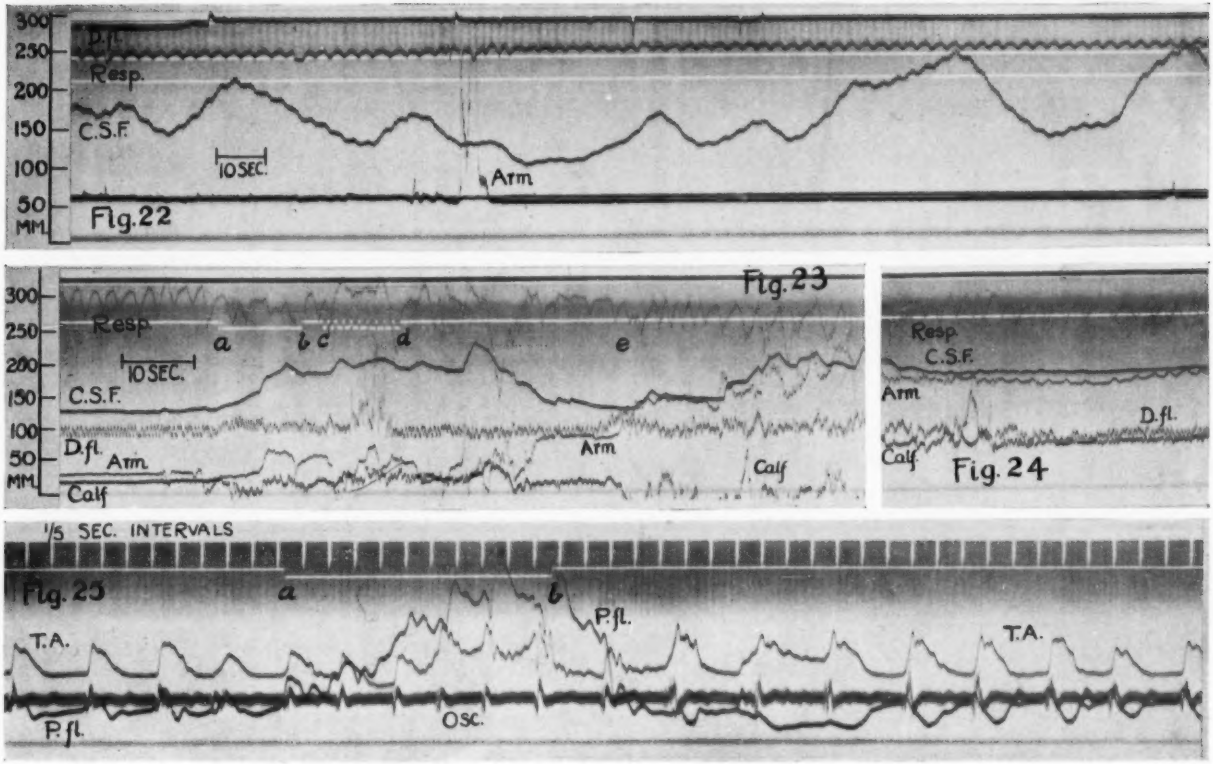

300
250
200
150
100
50
MM.
P.fl.
Resp.
C.S.F.
10 SEC.
Arm
Fig. 22
Fig. 23
Resp.
a
b c
d
e
10 SEC.
C.S.F.
D.fl.
Arm
Arm
Calf
Calf
Resp.
C.S.F.
Arm
D.fl.
Calf
Fig. 24
1/5 SEC. INTERVALS
Fig. 25
a
b
T.A.
P.fl.
T.A.
P.fl.
OSC.

PLATE V.

and the left side of the face. With this type of attack, which lasted five to 10 minutes, there was no loss of consciousness. During these attacks all the fingers of the left hand were painful, as if ' broken off at the knuckles,' and following the sudden cessation of the attack, in all the parts involved she ' gradually felt the fingers coming back.' Occasionally a similar sensation was felt in the toes of her left foot. The right side was never affected. Commencing in July, 1933, there was an interval of four months without an attack. At the end of this time similar attacks recommenced at weekly intervals. In the three months before admission the left leg and foot were noticed to show irregular, almost constant, twitching, and the limb was felt to be weak on voluntary movement. Intermittent spread of the twitching from the left leg to the thigh, sometimes to the left upper arm, and more rarely to both left upper and lower limbs and the face, occurred. With this spread was associated a continuous drawing pain and numbness in the left leg as high as the knee and the left leg felt ' as though broken off at the ankle.' Medicinal treatment had been regular for three years.

DESCRIPTION OF PLATE V

Plate V. Fig. 22 (Case V).—C.S.F. pressure, respiration and movements of foot, arm and face, as in figs. 17 and 18. To show a very small myoclonic convulsion chiefly affecting the arm, during partial emergence from sleep.

Fig. 23 (Case VI).—Record of plantar stimulation and resultant myoclonic activity with onset of myoclonic convulsion after a further interval. Beside the cerebrospinal fluid pressure (C.S.F.) there is recorded movement of the chest (Resp.), dorsiflexion of the foot (D.fl.) with upward movement for dorsiflexion, flexion of elbow (Arm) recorded by balloon over belly of biceps, and contraction of calf muscles (Calf) by small balloon adherent to gastrocnemius. At the beginning of the tracing a regular series of small movements of dorsiflexion of the foot are occurring ; they had been present for some hours. As will be seen in later records their irregularity is at times interrupted by small pauses, at times by greater fusion of the twitches. In the interval indicated by the fall of the white signal line from *a* to *b* the sole of the left foot was stimulated with a blunt object causing some further tonic dorsiflexion of the foot and some general movement of the patient, with jerking of the arm record and irregularity of the respiratory record with rise of cerebrospinal fluid pressure. From *c* to *d* violent clonic movements of the foot, trunk and arm occurred which appeared to the observers to be involuntary in origin. These movements and the resulting irregularity in respiration rapidly settled down, but at *e* the patient commenced to have a major convulsion during which she was unable to speak. This lasted some minutes and the resulting disturbance declined with gradual lessening of myoclonic twitchings, the termination of which is recorded in fig. 24.

Fig. 24 (Case VI).—The termination of the attack commencing in fig. 23. The alteration in the relative position of the movement records is due to change in posture of the arm and leg.

Fig. 25 (Case VI).—Record taken at faster rate to show the nature of the twitchings occurring at this time in both tibialis anticus and the calf muscles. Each twitch was of the nature of a slight plantar flexion of the foot passing immediately into dorsiflexion at the ankle with plantar flexion of the toes. The speed of the record is here increased so that the vertical white lines marking one-fifth seconds are here widely spaced. The horizontal thick record is that of the beam of a Matthews oscillograph (Osc.), which records the action currents in tibialis anticus. The upward displacement of the upper record (T.A.) reveals contraction of tibialis anticus, upward displacement of the lower record (D.fl.) reveals plantar flexion at the ankle. Each twitch of contraction in tibialis anticus is seen to be accompanied by an initial asynchronous series of small action currents. For the duration of the fall of the signal between *a* and *b* the patient is directed voluntarily to plantarflex the foot.

On examination no abnormality was found in the cranial nerves or the upper limbs. There was slight spasticity in the left lower limb with some impairment of muscle power at all joints. The right lower limb was unaffected. The tendon jerks were increased in all limbs and more brisk in the left lower limb than in the right. The abdominal reflexes were equal and active, the left plantar response extensor, the right flexor. Sensation was normal except for a slight impairment of vibration sense over the left tibia. At times the peroneal muscles in the left lower limb, at others the flexors of the toes only, showed a fairly regular rapid series of twitches, which would continue for many hours. At irregular intervals the twitching would become more rapid and of wider amplitude and spread in the course of some seconds to involve the left thigh, left side of abdomen, left upper limb, and often the left side of the face. After two to three minutes the twitching would cease in the left side of the face and the left upper limb to remain in the toes or foot as before. Just after the subsidence of an attack pricking of any part of the left lower limb would again evoke spread to the left upper limb and face. On some occasions, but not in all, handling of the left lower limb during a quiet interval would be followed by a full spread of the attack. The same effect could be evoked by pin-pricks in this limb. There was no loss of consciousness in the attack. A right fronto-temporal surgical exploration revealed no surface change in the cerebral cortex. The attacks were continuing as before when the patient was last under observation early in June, 1934.

Analysis of attacks recorded on March 26 and May 7, 1934.—The cerebrospinal fluid pressure in this patient was 210 mm., fluctuating between 262 and 280 mm. as she fell asleep. During a period of observation of just over three hours there was continued irregular twitching of the tibialis anticus. For the greater part of the time no other muscles participated in this irregular twitching movement. The twitches were small and showed a general regularity in amplitude. When recorded by a balloon over the tendon of the muscle, connected to a small tambour manometer of high vibration frequency, they were found to take the form of groups of muscle twitches at times in small series of three to five, at times in partly fused pairs and often for long periods in a rhythm of single slightly longer waves at the rate of about two a second (fig. 23). There was no corresponding change in the cerebrospinal fluid pressure. At intervals of one to two hours the rate of twitching accelerated in tibialis anticus and at the same time twitching in the calf muscles, more proximal parts of the left lower limb and in the left arm occurred. The transition between the local attack and its generalization was gradual, as from *d* to *e* in fig. 23. The spread of the attack occurred in steps rather than in a gradual march. It was noticed that twitching of the calf muscles of minor kind would often accompany the twitching in the pretibial muscles for periods of several minutes without further spread of the attack. These continued twitches would often persist for many hours. On another occasion it was noticed that the twitching remained confined to the posterior tibial group of muscles alone for long periods. During the recorded observations the twitchings did not completely cease during sleep, but this was often observed in the ward.

The insertion of the lumbar puncture needle, the elicitation of a left plantar response, and pricking the left lower limb were observed to be followed

by a generalized attack, but only when such an attack had not occurred for a long period. The result of scratching the sole of the left foot and the subsequent development of a widespread attack is shown in fig. 23. The plantar stimulation here evoked irregular twitching of the upper limb, and twitching continued in the calf, trunk and upper limb for some 20 seconds. With the irregular trunk movements the cerebrospinal fluid pressure rose unevenly and with their lessening fell to its original level. Within a few seconds twitching again occurred in all the parts concerned, becoming of very rapid vibratory kind: the attack having thus commenced continued irregularly for four minutes. The initial violence of the attack subsided somewhat after the first two minutes, and as respiration became more regular the cerebrospinal fluid pressure subsided more gradually, although the tension remained higher than before for many more minutes (fig. 24). There was no apparent loss of consciousness during the attacks. In this patient during a quiescent interval acti· › hyperpnœa for a period of three and a half minutes did not evoke spread of the twitching which was at the time confined to tibialis anticus. A plantar response one minute later was followed by involvement of all the muscles of the lower limb in the twitching. A period of hyperpnœa lowered the cerebrospinal fluid pressure from 190 to 170 mm., and recovery of pressure was delayed for a considerable interval.

On May 7, 1934, the twitching in tibialis anticus was recorded electromyographically together with mechanical records of the contraction of this muscle and of the calf muscles. The tetanic nature of each twitch and the way in which the twitching summated with voluntary movement and ankle clonus are discussed below.

DISCUSSION

It is evident that the recorded convulsions in these six patients differ considerably from patient to patient.

We were fortunate in having for these observations a wide range of phenomena, from the rapidly repetitive and very localized discharge of epilepsia partialis continua in Case VI to major attacks of sufficient violence to require the greatest perseverance in securing records, as in Case II. All patients with the exception of Case V had at one time or another suffered from major convulsions, and we believe that the phenomena which they exhibit may be taken as fully representative of the effects of that process which underlies the symptom of epilepsy, and to which for brevity we shall refer as the epileptic process. We present reasons for the belief that this process was present in Case V. The myoclonus in these cases was of the more usual variety encountered in epilepsy, without familial characteristics.

It may be remarked in passing that the present cases serve to throw into prominence the artificiality of any very close identification of any particular variety of epilepsy with any particular variety of provocative pathology. The only rigid classification of epilepsy into organic and idiopathic categories is one of morbid anatomy.

The Cerebrospinal Fluid Pressure

All observers are agreed that the cerebrospinal fluid pressure is increased during major epileptic convulsions, and pressures of 450 mm. (Redlich and Pötzl [24]), 500 mm. (Dalma[4]), 700 mm. (Lennox and Cobb [24]), and 870 mm. (Nawratzki and Arndt [22]) have been noted. Such changes in pressure might reasonably be expected as direct effects of the violent movements of major attacks. Ebaugh and Stevenson,[7] by means of a tambour attached over a decompression opening in the skull, have observed a lowering of intracranial pressure for an interval of four and a half hours, covering three attacks of petit mal in one patient, while Elsberg [8] has observed a rise of blood pressure and increased bulging of a decompression opening for six to eight hours before the Jacksonian attacks of one patient. Macdonald and Cobb [18] have observed an initial fall of pressure preceding the onset of the convulsive stage in animals intoxicated with thujone. This initial fall of cerebrospinal fluid pressure, possibly related to the sudden pallor of the cerebral cortex occasionally observed by surgeons to precede a convulsion (Kennedy,[15] Leriche [17]), accordingly has some more direct pathological interest.

It is evident from our records that abrupt change in the cerebrospinal fluid pressure may occur very early in the course of an epileptic fit. This change, which may be a fall or a rise, tends to a regular form in consecutive attacks in any one of the patients examined, and is much more obvious in Cases I, III, IV and VI than in Cases II and V.

Of the numerous factors which affect the cerebrospinal fluid pressure, the venous pressure is conspicuous in its rapidity and constancy of effect, characteristics which find common employment in the Queckenstedt and Ayer-Toby tests. The change in pressure engendered by light jugular compression as used in these tests is at first abrupt and rapid and then gradually reaches a steady plateau (fig. 4). The increase varies in height from patient to patient and according as the right or left jugular vein is compressed. Its mechanism may properly be related to distension of the cerebral veins, and its variability in extent to the anatomical variations of the drainage to each lateral sinus. The final plateau in this view indicates the full congestion of the cerebral veins drained by the sinus which is compressed.

The rudimentary valves of the jugular veins are well known to be incapable of obstruction of back pressure in this system so that from the right auricle pressure effects are directly transmitted to the cerebral veins. In such manner the rapid change in cerebrospinal fluid pressure early in a cough, as seen in fig. 9, receives explanation as a direct transmission of a wave of increased venous pressure from the thorax to the cerebral and spinal veins. When the patient is lying almost horizontal the cerebral veins are slightly distended so that not only is an increase of pressure transmitted from the thorax to the cerebrospinal fluid, but also any diminution of intrathoracic pressure. In this way the initial deep inspiration of the fit in Case IV, well seen in figs. 13, 14 and 15, accounts fully for the remarkably constant initial sharp

fall of cerebrospinal fluid pressure in the attacks of this patient. So, too, close scrutiny of the record of chest movement in Cases I and III reveals a small irregularity, a small forced expiration, which accounts for the initial rise in these patients. The subsequent rise in the cerebrospinal fluid pressure is related to the period of apnœa, where the chest is fixed in expiratory spasm. The subsequent decline mirrors the gradual resumption of normal respiration which may be early after the termination of the attack as in fig. 1, or much delayed, as in figs. 12 and 24.

Rapid deep breathing causes a fall of cerebrospinal fluid pressure, as shown by Stevenson, Christensen and Wortis.[30] No doubt the rapid fall in pressure to a low level following some fits (fig. 1) is the result of hyperventilation in the last stages of the attack.

In Cases II and IV, in which maintained change of pressure did not usually occur, the thorax and abdomen, though involved by myoclonic twitching, did not show inspiratory spasms (figs. 6, 17, 19). Increased rapidity of respiration has but little effect, as at *b* in fig. 12, and here later turning of the head with tonic contraction of the chest brings about a delayed rise.

The change in cerebrospinal fluid pressure in these recorded cases therefore reflects both the degree of expiratory spasm of the thorax and the time of that spasm in relation to the other phenomena of the attack. The relative constancy of form of the pressure change in any one patient is but evidence of the regularity of march of events in the attacks of that particular patient. The exact commencement of the convulsion is difficult to define. From records such as figs. 12, 13 and 14 it is evident that some movement of the chest may precede the onset of convulsive phenomena in the limbs. Pollock and Treadway [23] found changes in respiration preceding the convulsion in 12 out of 44 cases investigated, in the nature of an apnœa lasting 16 to 30 seconds, accompanied by fall in blood pressure. A similar affection of respiration possibly accounts fully for the fall in blood and cerebrospinal fluid pressure preceding thujone convulsions in animals.[18] This change in respiration appears to us more properly regarded as an early convulsive phenomenon. In the preconvulsive stage we have observed a constant change in cerebrospinal fluid pressure in only one patient (Case V), a slow fall of pressure over many minutes, culminating in the onset of the convulsion. The mechanism of this type of preliminary change is discussed below.

The Effect of Sleep

Brodmann [1] and Shephard,[26, 27] by means of a tambour applied over the site of cranial decompression openings, observed a rise of intracranial pressure during sleep, in contrast with the fall that Mosso [20] had observed by a similar method. Stevenson, Christenson and Wortis,[30] by continuous records of cerebrospinal fluid pressure by lumbar puncture, observed a rise of pressure during sleep. The amount of rise is not stated. In our Case V rise of pressure, with superimposed fluctuations, during sleep and drowsy

states assumed most remarkable proportions (figs. 18 and 22), so that large waves of 100–150 mm. height and lasting over some minutes occurred. Such fluctuations were noted by Brodmann and Shephard in trephine openings and in drowsy states by Stevenson, Christensen and Wortis in the pressure by lumbar puncture. They have been identified with the well-known Traube-Hering waves in the venous and arterial pressure,[11, 26, 27] and their appearance in the cerebrospinal fluid is presumably a direct transmission via the cerebral veins. The great depth of fluctuation in Case V is possibly related to the presence of hydrocephalus as revealed by postmortem finding. In Cases I and II the fluctuations were slighter in degree but preserved the same characteristics (fig. 5).

In view of the increase of vascularity of the cerebrum which is necessarily entailed in these rises, it does not appear to us possible to maintain that sleep is related to cerebral anæmia. We have not observed any fluctuation in rate or amplitude of respiration accompanying these waves in the cerebrospinal fluid. Their origin is therefore related to some parallel fluctuation in pressure on the right side of the heart. Any explanation by periodic fatigue of the sympathetic centres[30] or vasomotor centre[11] appears to find more difficulty in explanation than fluctuations of tone in the trunk and limb musculature.

Whatever be the true explanation of this phenomenon the slow fall of cerebrospinal fluid pressure preceding the attacks in Case V is clearly the result of transition from sleeping to wakeful state (figs. 18–21). This transition no doubt plays a rôle in the circumstances predisposing to the onset of the fit, but cannot be termed an integral part of the attack.

'Reflex' Epilepsy

In four of the patients subjected to examination clinical observation of the frequent appearance of an attack shortly after some unusual stimulus had suggested the presence of 'reflex' epilepsy. This phenomenon, of which examples are described by Jackson,[13] Gowers,[9] Wilson,[32] and others, occurs in varying degrees. It has been described in very localized form such as in the case of Jackson, where a touch only on the face or head regularly elicited an attack. More commonly cases have been described where attacks may be regularly evoked by a sound. In both varieties the element of surprise appears to be necessary. Jackson states of his case that 'a fit would not occur if he knew he was going to be touched.' The provocative noise must be 'a "startling" noise' (Gowers[9]), and of such a case Wilson[32] notes '. . . aura and subsequent fit never arise "spontaneously" but always as a sequence to a sudden extrinsic stimulus of any kind provided it is sufficient to startle him.' To one of us a young man subject to fits immediately after any loud unexpected noise once explained that he had had an attack when a piece of paper lying nearby was unexpectedly turned over by a gust of wind, *without any noise*. It is also not uncommon to find that handling of

the predominantly affected limb in a patient liable to focal epilepsy will occasionally set up an attack, as described by Gowers.[9] In this variety of 'reflex' epilepsy, as we ourselves have observed, the manœuvre may be repeated many times without eliciting an attack, and it is only when sufficient interval has elapsed since a previous attack that the association can be demonstrated. This phenomenon has not in our experience been associated with 'acoustico-motor' epilepsy, but was seen by one of us in a patient previously liable to a right-sided attack when the right ear was touched.[6]

In the present series of cases the patient of Case IV during clinical examinations had seemed to commence an attack immediately following any stimulus to the affected limbs. During the periods that records were made the attacks occurred with remarkable regularity, and as has been explained in the protocol the records show no significant change of interval when a limb had been stimulated. The brevity of the intervals at the time of recording may have resulted in the disappearance of the phenomenon, but from our records we have to regard the apparent association of stimulus and fit in this patient as probably a coincidence. In Case II attacks were more regularly elicited by noise or other surprise, and in Case VI by handling or stimulation of the affected lower limb. The attack arose at an interval from the stimulus (figs. 7, 17, 23) and differed in no way from those which arose spontaneously. The interval between stimulus and attack was, however, most interesting. In both patients the stimulus aroused an immediate outburst of myoclonic twitching. Such jerking is well known clinically to be provoked by surprise or start, as the studies of Pierce Clark [2] and Muskens [21] especially emphasize. Stimuli such as a scratch to the sole of the foot may be expected to increase local reflex excitability, and we would attribute the enhancement of the twitching in the foot by such a stimulus in Case VI (fig. 23) to such a process. If the cortical epileptic discharge may be regarded as having widespread subthreshold effects on spinal neurones other than those provoked to actual discharge, the 'reflex' enhancement becomes the summation of two stimuli, one reflex, one epileptic, on the common final path.

In Case V the occasional appearance of myoclonus during the troughs of the waves of cerebrospinal fluid pressure (fig. 22) may be taken as evidence of direct association of the waking state and such epilepsy. It is therefore further possible to interpret the epileptogenous effect of such a stimulus as pricking in the same patient (fig. 17) as an indirect result mediated by greater wakefulness. The long delay in appearance of the fit and the falling cerebrospinal fluid pressure document the process of awakening, to which the term 'reflex' cannot with accuracy be applied.

It appears to us that the process of myoclonic stimulation in waking states, such as recorded in fig. 7, has only quantitative difference. The patient is awakened from a state of partial wakefulness to one of greater wakefulness.

To align the phenomena of 'reflex' epilepsy with the known charac-

teristics of spinal reflexes it appears to us to be necessary to abandon the notion that the fundamental epileptic process is in some way part of a reflex arc and to substitute the alternative hypothesis that the fundamental epileptic process is continuous and widespread, and in its lesser intensities productive of outward effect only by coalition with other intense nervous processes. In this view the effect of an added stimulus is related solely to its power to cause a sudden increase in excitability in the patient concerned. In Case VI the spasticity and heightened flexion reflex in the left lower limb as a result of a cerebral lesion not only enable the enhancement of attacks by stimuli to that limb, but possibly may be held to contain the explanation of epilepsia partialis continua in that limb for many months. The early pallor with fall of blood pressure in some generalized epileptic attacks, and particularly (though not constantly) in petit mal, and the early apnœa we have recorded, may in this view be but the first evidence of the rise of generalized epileptic excitation, reflected in nervous mechanisms already active, and hence more sensitive in revealing change. It is immaterial that the neural levels of these mechanisms are the lowest, for the rising epileptic excitation is passed on to all levels, or conversely a cortical representation may be urged for all.[9]

Sudden 'starts' can be elicited from the thalamic animal by a loud sound. Muskens [21] claims that similar starts have a reflex mechanism in the pons, and identifies them with the myoclonic twitch of epilepsy. He has widely investigated the enhancement of this reflex by monobromide of camphor. We would urge that the start reflex be regarded as a true reflex distinct from the epileptic state. Epileptic myoclonic twitching exists in patients in whom the reflex start to surprising stimuli is not present, and in them auditory stimuli do not appear to provoke attacks. Where the start reflex is readily obtained in an epileptic patient, as in our Case II, a sudden jerk in the flexors of the limbs is the immediate consequence of an unexpected sound. The jerk or 'start' facilitates an outburst of myoclonic twitching, and whether or not an epileptic fit develops appears to depend on the coincident condition of the 'epileptic state.' In this way the effects of wakefulness, spinal reflex, and reflex 'start' may be brought into the category of facilitation at the final common pathway. Whether the site of convergence of these effects be cortical, or pontine, or spinal probably differs from case to case. On general grounds it is our belief the common site is cortical, some reasons for which will be discussed below.

The Incoordination of the Epileptic Discharge

It has been maintained as a result of cinematographic analysis of epileptic convulsions (Stenvers [29]) that the movements are sequences of coordinated single movements such as the neck reflexes of Magnus. Martin [19] describes the movements of the legs as spinal reflexes similar to the walking reflexes of experimental animals. Such movements should be highly organised, so

that there is essential harmony of contraction in each phase, and they should be completely integrated.

In our records of epileptic myoclonus and the phenomenon of epilepsia partialis continua there is frequent evidence of complete irregularity both in time and place, and we feel that this lack of organization is of importance not only in these minor convulsive phenomena but in all degrees of epileptic convulsion. The biting of the tongue is clear expression of this fundamental incoordination. In this we find full accord with the definition of epileptic discharge as a 'discord of movement' (Gordon Holmes [10]), and the statement of Kinnier Wilson [32] that the outward 'caricatured movements in no way resemble those called voluntary.' The sole coordinated motor discharges of epilepsy are the behaviour phenomena of the aura and of automatism which are not proper to the convulsive stage with which we are here alone concerned.

The essential irregularity of the myoclonic convulsion is seen in figs. 8 and 16, where various groups of muscles enter haphazard contraction in irregular time relation. Records of the twitching in Case VI (figs. 24–29) appear of particular interest in this connexion, for here long continued cortical discharge excited flexors and extensors concurrently, and brought a twinge of pain into each momentary paroxysm. The foot was contorted in meaningless fashion. In the records of major attacks in other patients the convulsion of the chest not only made abrupt cessation of respiration, but its incoordination with the movements of the glottis produced the 'epileptic cry.'

Case VI was also particularly suitable for the exploration of the relationship between voluntary and other movement and the intermittent paroxysms. A Matthews oscillograph gave us particularly clear records of the nature of the discharge. In the tibialis anticus this took the form of outbursts, each lasting usually one-fortieth of a second, and composed of six to 11 rapid galvanometer vibrations, each corresponding to a volley of impulses (fig. 25). The resulting twitch of the muscle thus appears to be in the nature of a brief motor tetanus. The intervals between the outbursts are 'silent,' and no discharge reaches the muscle. This discharge differs from reflex spinal clonus in the scattering of the discharged impulses, for in the latter there is synchronous or almost synchronous discharge of one volley for each wave of muscular contraction.

Voluntary movement had no effect upon this discharge. During voluntary plantar flexion the discharge continued as before (fig. 25). Voluntary dorsiflexion of the foot added a normal continuous series of nerve impulses in rapid sequence to the epileptic discharge, so that if the movement were powerful the epileptic discharge was almost swamped (fig. 27), while with weaker efforts the epileptic impulses were less covered (fig. 28).

At intervals each outburst of discharge increased in duration and the intervals lengthened. This change occurred either as the occasional appearance of an isolated longer outburst of one-tenth second duration, or by the

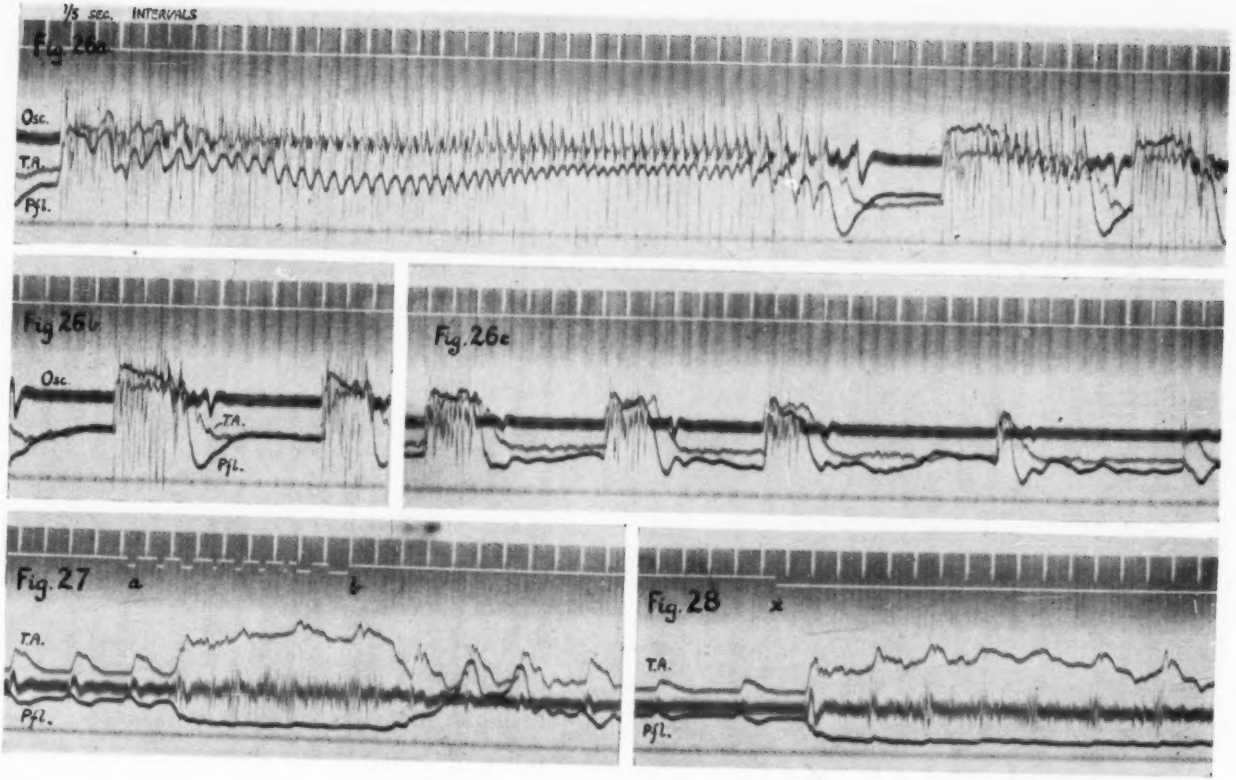

1/5 SEC. INTERVALS
Fig. 26a.
Osc.
T.A.
Pfl.
Fig. 26b
Osc.
T.A.
Pfl.
Fig. 26c
Fig. 27 a b
T.A.
Pfl.
Fig. 28 x
T.A.
Pfl.

PLATE VI.

progressive lengthening of discharge to separate continuous outbursts of tetanic discharge, each lasting up to 10 seconds. An example of the latter type is shown in fig. 26a, and the progressive shortening which characterized the return to the more usual rhythm. The prolonged outburst is seen to be essentially clonic in nature, with frequency increasing from eight to 13 beats a second. The corresponding muscular contraction is a partially fused tetanus, and still involves both the opposing dorsiflexors and plantar flexors of the ankle. In both brief and prolonged bouts of discharge a contraction of the plantar flexors of the ankle preceded the contraction of tibialis anticus by a brief interval, tending to lessen as the contraction of tibialis anticus increased. Following a series of such spasms of increased duration the usual brief attacks either were much diminished in amplitude or disappeared entirely for some minutes. In the major attack in this patient, shown in figs. 23 and 24, this type of discharge does not appear, though the duration of each twitch is longer during the attack than before, especially in the partial attack elicited by stimulation of the sole of the foot (fig. 23, *c–d*). We do not regard such maintained clonic outbursts as different from the more irregular variety recorded in spontaneous major attacks. Both were invariably accompanied by spread of the epilepsy to other muscles. Prolongation of discharge by change in rhythm is therefore one aspect of increasing activity of the epileptic process.

It was readily possible in this patient to elicit ankle clonus in the affected limb. In fig. 29 is shown the effect of combining the local reflex rhythm of ankle clonus with the rhythmical epileptic discharge both of the discrete brief type and of the more prolonged type. The transition in type of discharge is here possibly in part due to the handling of the limb, such transitions being often found under these circumstances. As with voluntary movement, the rhythmical cortical discharge continued throughout the duration of induced ankle clonus, and affected the latter in two ways. The heightened tone of the calf muscles was diminished during each twitch of tibialis anticus, and the rhythm of the ankle clonus was reset by each epileptic outburst

PLATE VI.

DESCRIPTION OF PLATE VI

Plate VI. Fig. 26 (Case VI).—Records as in fig. 25 to show the nature of long-maintained clonic attacks which occasionally arose spontaneously in this patient. Fig. 26a is an example of a long-maintained tonic attack, and the gradual resumption of the more usual smaller attack at the end of fig. 26c. Fig. 26b is the direct continuation of fig. 26a, while fig. 26c followed fig. 26b at an interval of $3\frac{2}{3}$ seconds. The single slow wave in the electromyogram after the termination of each attack is due to passive movement of the galvanometer leads.

Fig. 27 (Case VI).—As figs. 25 and 26. To show the effect of strong voluntary dorsiflexion of the foot which the patient was ordered to perform in the duration of the interruptions of the signal between *a* and *b*.

Fig. 28 (Case VI).—As fig. 27, a few seconds later in the experiment. At *x* the patient was ordered to make a slight voluntary dorsiflexion of the foot.

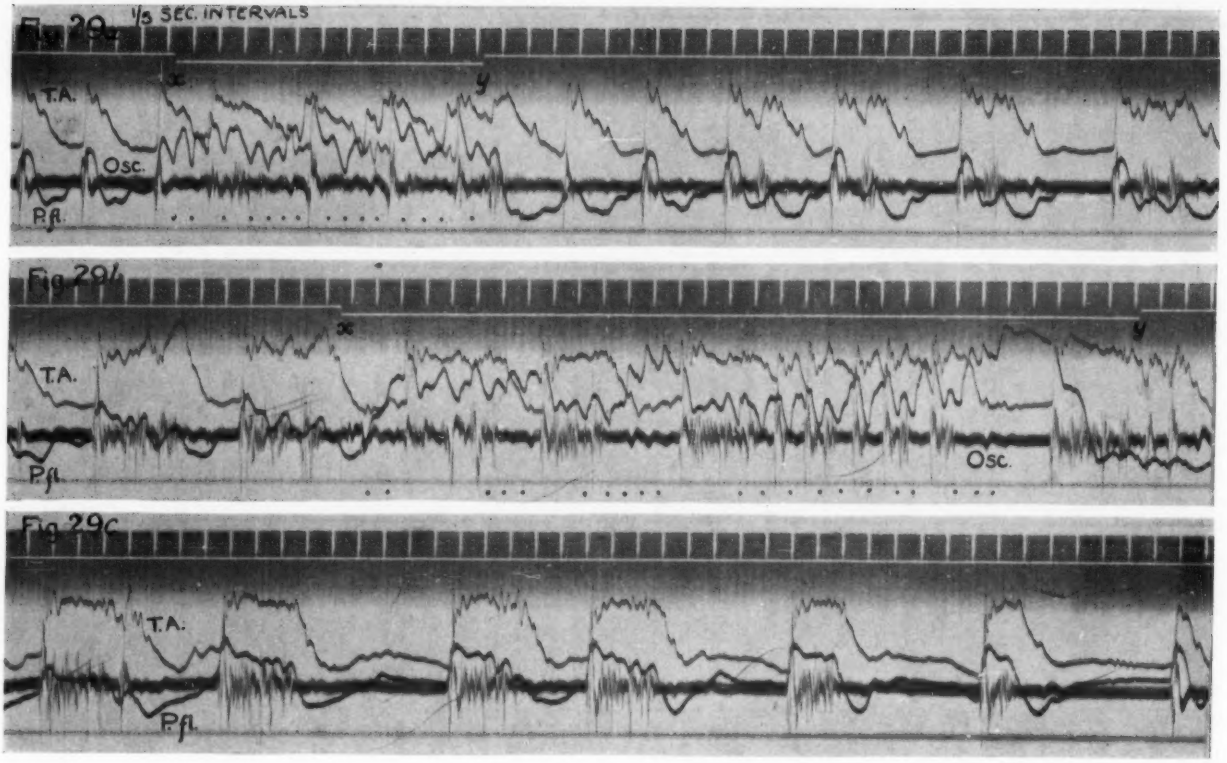


Fig 29a
1/5 SEC. INTERVALS
T.A.
Osc.
P.fl.
x
y
Fig 29b
T.A.
P.fl.
x
y
Osc.
Fig 29c
T.A.
P.fl.

PLATE VII.

(fig. 29a). When the epileptic discharge is more prolonged the rhythm of ankle clonus apparently combines with the rhythm of cortical clonus, and in tibialis anticus the epileptic action currents assume combinations between 'cortical' rhythm and the reflex rhythm of its opposing muscular group (fig. 29b).

The cortical discharge of epilepsy is therefore independent both of voluntary movement and of spinal reflexes, though both combinations sum their effects in the final common pathway. There being no certain evidence that the epileptic discharge is transmitted by pathways other than the pyramidal tract, we may provisionally assume that the locus of convergence of voluntary and epileptic innervation is on the dendritic expansions of the Betz cells in the motor cortex.

In Cases II and V myoclonic twitching actuated each muscle for short periods in brief attacks, and in records of movement such as fig. 16 the character of the discharge in any one muscle differs not at all from that occurring more regularly in the muscles of Case VI. In the former patients the epileptic process has multiple foci of fluctuation; in the latter a single focus in the leg area varies in the spread of its effect. Whether the convulsion be particulate as in myoclonus, or diffuse as in the major attack, incoordination of the cortical discharge is its essential feature. It reflects a global stimulation of a cortical area foreign to the selective niceties of voluntary movement.

Some degree of concurrent contraction of the opposing flexor and extensor muscle groups at a joint is not uncommon in the course of spinal reflexes. The phenomenon most commonly arises in the antagonism between two contrary reflexes of similar potency and has explanation in the process of 'double reciprocal innervation.'[28] Its analysis in the laboratory animal demonstrated clearly that here the spinal neural mechanism preserves a regulated relative balance between opponents, so that the law of reciprocal innervation is not in fact upset. In more complex posturing variable degrees of concurrent innervation of opponents are also found, and for reflex movement

PLATE VII.

DESCRIPTION OF PLATE VII

Plate VII. Fig. 29 (Case VI).—Records of contraction of tibialis anticus (T.A.) and plantar flexion of the foot (P.fl.) with electromyogram of tibialis anticus, as in figs. 25 to 28. The more usual small epileptic twitching had continued uninterrupted for some minutes before this record. In fig. 29a three such beats occur when at x an observer by passive dorsiflexion of the ankle elicits ankle clonus. The individual beats of the clonus are seen in the record P.fl. and the time of onset of each is further indicated by the black dots, which have been placed below the record. Following the termination of the ankle clonus at y the epileptic twitching continues with prolongation of the beats and the intervals between beats.

Record in fig. 29b commences after the lapse of some 10 seconds during which there was no material change in the discharge. At x ankle clonus is again elicited by an observer and is discontinued two-fifths sec. before y.

Fig. 29c shows the course of events in direct continuation of fig. 29b.

it is the invariable rule that for each moiety of contraction exerted in the prime movers there is an opposite inhibition of the opposing muscle centre. The more rapid the movement the more effective is the counter inhibition.

Voluntary movement in man possesses in its synergy readily verifiable co-contraction of antagonists, a feature which has been maintained by some to invalidate the hypothesis of reciprocal innervation. Synergic concurrent contraction has, however, another aspect. The difficulty in overcoming the spasm of cramp, or the burden of spasticity, by voluntary movement is sufficient disability to make it clear that the contraction of an antagonist most definitely requires limitation. It is a philosophical necessity to enquire whence this limitation, which ensures the precision and rapidity of voluntary movement, is derived. It appears to us to require the most exquisite final development of that process of regulation of antagonists which the principle of reciprocal innervation defines.

The convulsions of asphyxia or of intoxication by strychnine in the spinal animal are composed of alternations of rapid scratching, running, leaping, vomiting, each well coordinated and of regular though rapid rhythm.

Movements resulting from moderate electrical stimulation of a motor point in the cerebral cortex possess outward show of coordination, probably mediated by the spinal motor mechanism. Immediately the stimulus is removed a sudden damping of all discharge in all muscles is often followed by a powerful outburst of contraction unlike the primary movement. Alternating outbursts of this after-contraction form the well-known epilepsy resulting from cortical stimulation in animals, a phenomenon which at times marches from muscle to muscle. Examined myographically, the individual spasms of this 'clonus' are at times reciprocal in opposing muscles, but at others are concurrent, producing an incoordination so powerful that double reciprocal innervation no longer accounts for it.[3] This incoordination of the 'after-discharge' agrees with other evidence that the electrically produced movement is itself also the outcome of a mixture of effects in which outward coordination appears to be the added approximation of reciprocation by the spinal mechanism. However small the electrode, a stimulus which excites the motor cortex at all excites a mosaic of units of differing and opposing functional sign.[3] Clonic after-discharge is the rhythmical dissipation of incompatible residues.

Both in the records of epileptic clonus here described and in the epileptiform after-clonus from cortical stimulation the tetanic nature of each individual outburst, the complete silence between outbursts, and the lack of reciprocal coordination serve to identify the same underlying mechanism of production. The clonic spinal stretch reflex (spinal clonus) is by complete contrast a series of synchronous volleys of single nerve impulses. These differences between convulsive clonus and spinal clonus render it extremely unlikely that the former is a release of spinal reflexes by the removal of

cortical inhibition. We have already remarked that lack of reciprocal innervation in convulsive discharge has only a quantitative difference from that which is traceable in the result of unipolar stimulation of the cortex. Both represent the global stimulation of a microscopical mosaic. Conversely the property of activation of single movements, or even single muscles, is common to both, and reflects the stimulation of relative concentrations of units of like function from a general anatomical pattern unlike any other in the nervous system. A movement limited to a single joint (cf. Cases V and VI) stamps its origin from the motor cortex as much for epilepsy as for electrical stimulation. In the myoclonic variety of epilepsy such isolated movements (figs. 8 and 16) may be watched for hours on end, and their local character completely denies any pontobulbar localization of their origin.

We therefore conclude that the discharge of an epileptic convulsion may be more truly likened to a discord of the units of movement, a discord without component melodies, so that it appears not strictly accurate to regard a convulsive movement as a ' contention of very many of the patient's familiar normal movements.' [14] The natural organization of voluntary movement has a selective mechanism which the convulsion ignores.

The Periodicity of Seizures

Periodicity of the motor discharge of epilepsy is present in many degrees. Not only is it found in the recurring twitches of epilepsia partialis continua and in myoclonus, but it is also present as the rapid clonic discharge which makes up longer outbursts. Besides these rhythmical phenomena in the single convulsion there has also long been recognized a tendency in some patients for fits to occur in groups, each group of as many as 20 fits being separated from similar groups by regular intervals (Gowers [9]). Status epilepticus occurs most usually as an isolated event, but in Cases II and IV the periodicity of its development was particularly well defined. In the former the frequency of attacks in three such outbursts at intervals of nine months and eight months is plotted in fig. 30. Although the phenomenon rarely occurs in such perfect form, minor variants, such as is outlined in the history of Case III, are not infrequently encountered. The phenomenon is as much a property of ' idiopathic ' epilepsy as of epilepsy of organic origin (Case IV). When the number of attacks from day to day in Case II are charted the regularity of accrescence and decrescence in number of attacks is even more striking, for, although the amount of treatment had been increased shortly before the peak of the attack, the smoothness of rise and decline, in spite of abrupt changes in treatment, gives evidence of a process of fluctuation not related to treatment. Portion of such a decline is also seen in the figures given for the frequency of observed attacks in Case III. Though at first glance the actual turn or peak of the graph appears wholly referable to the intensity of administration of sedatives in various forms, this interpretation fails to account for the absence of effect of heavy dosage in the

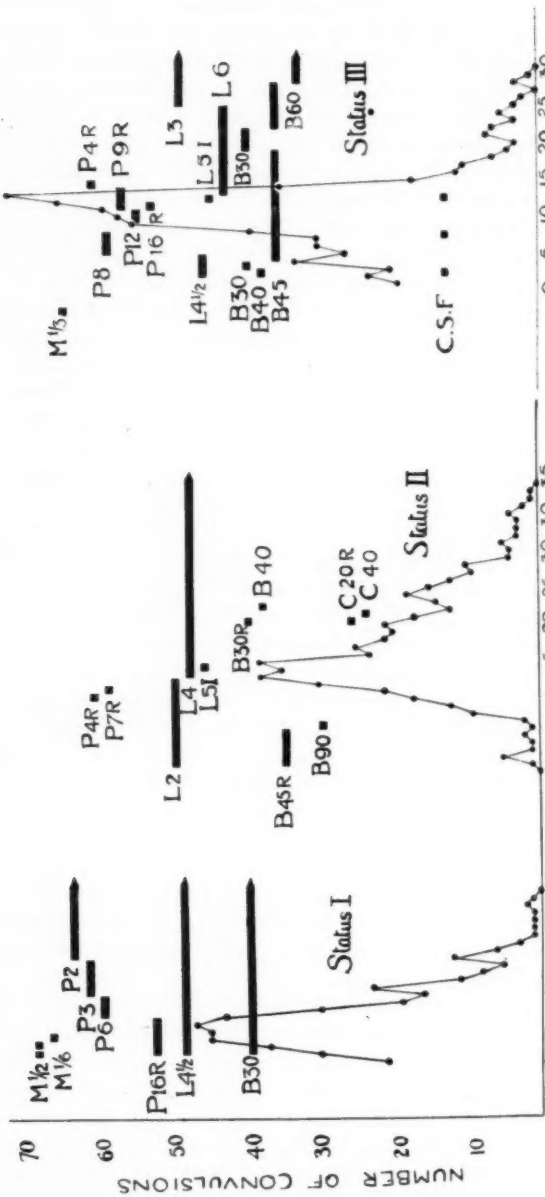

NUMBER OF CONVULSIONS
70
60
50
40
30
20
10
0 5 10 15 20 25
DAY OF OBSERVATION
0 5 10 15 20 25 30 35
0 5 10 15 20 25 30
Status I
Status II
Status III
M½
M⅙
P2
P3
P6
P16R
L4½
B30
P4R
P7R
L2
L4
L5I
B45R
B90
B30R
B40
C20R
C40
M⅓
P8
P12
P16R
P4R
P9R
L4½
L5I
L3
L6
B30
B30
B40
B45
B60
C.S.F

Fig. 30.

early stages in status III (fig. 30), or the final termination in diminishing series of few attacks in all three states. Indeed the initiation of a succession of attacks during regular treatment is strong evidence of some fluctuation in the underlying process. The form of the curves finds more ample interpretation as the expression of a natural evolution and involution deeper than the effect of treatment. Our Cases II, III and IV presented each the well-known transitory postepileptic hemiplegia of Todd [31] and Robertson.[25] In this condition the inevitably recurring attacks continue in limbs which may be early completely paralysed for all voluntary movement. In the intervals between convulsions they lie flaccid and inert. Though we have not made records of attacks in the same limbs before and after the appearance of this exhaustion, we have observed, especially in Case II, no essential difference apart from a shortening of duration of attack, a fact which is most difficult to reconcile with any of the theories of epilepsy as a reflex, or 'short circuiting,' or removal of inhibition, or indeed the explosive theory, all so fully discussed by Lennox and Cobb [16] in their monograph.

The time of onset and the time of complete development of postepileptic hemiplegia are not directly related to the commencement of decline in the numerical frequency of attacks, nor is any recurrence of fits referable to full recovery from the paralysis. The maintenance of the character of the attack during the paralysis further suggests that the phenomena have different anatomical loci. Neurones which cannot be voluntarily excited for periods of weeks continue to be available for the gross cortical discharge of the convulsions. The epileptic process is so situated that it may affect the mechanism of voluntary movement without impairing the spinal reflexes, for the tendon jerk, valuable indication of the state of the spinal reflex arcs, may remain unaffected. The flaccidity appears identical with that consequent upon lesions of the parietal cortex. Flaccidity of the affected limbs is by no means the rule, and variable degrees of spasticity may be present, even varying from one bout to another in the one patient, as in Case II. The phenomenon may in our experience affect cortical sensation as much as voluntary

---

Fig. 30.—Graphic representation of the number of attacks per day in Case II, in each of the three periods of status epilepticus in which this patient came under hospital observation. The number of attacks per day is recorded for each successive day covering 25 days from August 4, 1931 (status I), 35 days from May 18, 1932 (status II), and 30 days from January 2, 1933 (status III). Horizontal black lines indicate the duration of each of various forms of treatment. The lettering with each horizontal line records the form of treatment with the dosage in grains in each 24 hours; thus M. ½ represents morphia gr. ½ in 24 hours, P. represents paraldehyde with the number of drams by mouth in each 24 hours and where given per rectum the dosage is followed by the letter R. Phenobarbital is represented by the letter L, and where on two occasions 5 grains of phenobarbital sodium was given intravenously, this was represented by L 5 I. Bromide by mouth is represented by B with the number of grains in 24 hours. Chloral by the letter C with the dosage in grains in each 24 hours. On three occasions cerebrospinal fluid was withdrawn in quantities of 30, 36 and 30 c.c., represented by the squares after the letters C.S.F.

power (Cases II and IV). It is to be sharply distinguished on the one hand from the progressive hemiplegia resulting from an organic lesion as in Case I, and on the other from transitory loss of power when that occurs as a feature of isolated convulsions, as in Cases I and VI ('inhibitory epilepsy'). True postepileptic hemiplegia develops slowly after frequently repeated attacks. Those parts 'which have been first and most convulsed are those left paralysed' (Jackson [12]).

We have invoked the principle of subliminal (i.e. subthreshold) central effects to derive an explanation of 'reflex' epilepsy. In that case two processes, each alone ineffective, may combine to throw neurones on which they converge into discharge. It appears to be a logical corollary that any raising of threshold at the site of convergence may exclude weaker effects while still allowing the excitation by stronger. It is a general physiological principle that fatigue raises threshold, and the exhaustion of the outgoing neurones of the motor area by frequent epilepsy may reasonably be expected to result in a raised threshold to all synaptic convergence upon them. An epileptic convulsion may be likened to intense electrical stimulation of the motor area, so that it may remain effective when it has excluded voluntary movement by fatigue. A state of exhaustion which may persist for many days after the subsidence of the epileptic discharges appears a necessary postulate and was recognized by Todd, Robertson, Jackson, and others. Its relationship to the occurrence of an extensor plantar response for some minutes after each attack (cf. Cases II and III) is difficult to define. Provisionally it would appear necessary to regard both as aspects of the same phenomenon, but there is as yet no evidence that reflex changes of this kind are unrelated to persistent cortical after-effects other than exhaustion. In Case VI, myoclonic epilepsy in the right side disappeared with the onset of a permanent hemiplegia, but here widespread cortical disintegration was proceeding with great rapidity. An irregular high temperature accompanied the syphilitic encephalitis. In the other patients slight irregular temperatures up to 99·8° F. were common. We did not encounter in this series of cases the high temperatures of the more grave forms of status epilepticus.

The periodicity of epilepsy has two aspects, namely the interval between one attack and another, and the intervals between groups of attacks. The declining frequency of attacks as the grouped epilepsy passes off is often unaccompanied by exhaustion paralysis, just as there may be complete absence of impairment of voluntary innervation in any part of the intervals between attacks. These intervals must therefore be related to a variety of exhaustion in neurones other than the pyramidal tract and spinal motoneurones. The convulsive process overflows in discharge, while the means of its renewal, a fundamental epileptic process with longer rhythm of fluctuation, appears to be necessary to give rise to such periodic outbursts as grouped epilepsy or periodic status epilepticus.

Epilepsia partialis continua would appear to offer an example where

all the cortical processes of epilepsy are related to a well-defined group of cortical neurones, the motor area of the part concerned. In Case VI our analysis finds the pathway for motor innervation unaltered by the regularly recurring paroxysm, so that here with the simplest possible mechanism the course of the attack may be stated to originate beyond the mechanism of excitation and conduction in the Betz cells. There is no anatomical structure or physiological mechanism which can be identified with the locus of the fundamental epileptic state. It may be local or widespread in effect, and it may undergo fluctuation as a whole, or in most isolated subdivision such as we imagine to occur in myoclonic twitchings. Its effect on the motor cortex most closely resembles the diffuse stimulation of areas of variable extent.

While it may appear banal to reflect that the high proportion of fatal terminations of status epilepticus is evidence of exhaustion of the patient before exhaustion of the epileptogenous state, it nevertheless appears of the utmost importance that the rationale of treatment of the condition should not be the suppression of attacks at all costs, for in many patients this is not possible by any remedy other than a continuous general anæsthetic which appears to rest the epileptic process as much as, if not more than, the patient. A primary object of all treatment should be the adequate nourishment of the patient, combined with the maximum of sedative which is not otherwise injurious.

CONCLUSIONS

1. During epileptic convulsions the pressure of the cerebrospinal fluid shows changes reflecting passively the fluctuations of venous pressure arising chiefly from involvement of the thorax in the convulsion.

2. The cerebrospinal fluid pressure rises in sleep, and there is fluctuation in drowsy states. A slow fall of pressure preceding a convulsion is evidence only of an awakening of the patient.

3. Certain phenomena such as awakening, starts, or even local spinal reflexes, may enhance the tendency to epilepsy and thus appear to be the direct excitant of discharge. Such effects when they occur are examples only of the addition of one nervous excitation to another.

4. The convulsive stage of the epileptic attack is essentially an incoordinated discord resulting from the diffuse excitation of outgoing cortical neurones. Even in the most localized forms of epilepsy the essential epileptic process does not appear to be directly located in these neurones.

5. The exhaustion of nervous activity underlying postepileptic hemiplegia is related to threshold changes in the excitability of pyramidal neurones, whereas the intervals between attacks and the fluctuations in tendency to attacks are related to an unphysiological mechanism which has no defined anatomical locus.

We wish to acknowledge our indebtedness to the Medical Research Council for the use of myographic recording apparatus granted to one of us

(D. D.-B.), and to the various members of the Honorary Staff of the National Hospital for permission to investigate patients under their care.

REFERENCES

[1] Brodmann, K., *Jour. f. Psych. u. Neurol.*, 1902–3, **1**, 10.

[2] Clark, L. Pierce, *Jour. Nerv. Ment. Dis.*, 1919, **50**, 17.

[3] Cooper, S., and Denny-Brown, D., *Proc. Roy. Soc.*, 1927, **102B**, 222.

[4] Dalma, G., *Riv. sper. freniat.*, 1925, **49**, 371.

[5] Denny-Brown, D., and Robertson, E. Graeme, *Brain*, 1933, **56**, 149.

[6] Denny-Brown, D., *Proc. Roy. Soc. Med.*, 1929–30, **23** (Sect. Neurol.), 3.

[7] Ebaugh, F. G., and Stevenson, G. S., *Johns Hopkins Hosp. Bull.*, 1920, **31**, 440.

[8] Elsberg, C. A., *Surg. Gynæcol. Obst.*, 1916, **23**, 153.

[9] Gowers, W. R., *Epilepsy and other Convulsive Diseases*, 1881.

[10] Holmes, Gordon M., *Lancet*, 1927, **i**, 957.

[11] Howell, W. H., *Jour. Exper. Med.*, 1897, **2**, 313.

[12] Jackson, J. Hughlings, *West Riding Asylum Medical Reports*, 1876, **6**, 266, and *Selected Writings*, 1931, vol. I, 135.

[13] id., *Medical Society's Proc.*, 1886, **10**, and *Selected Writings*, 1931, vol. I, 362.

[14] id., Lumleian Lectures, *Brit. Med. Jour.*, 1890, **1**, 765, and *Selected Writings*, 1931, vol. I, 437.

[15] Kennedy, F., *Arch. Neurol. and Psychiat.*, 1923, **9**, 567.

[16] Lennox, W. G., and Cobb, S., *Epilepsy*, 1928.

[17] Leriche, R., *Presse méd.*, 1920, **28**, 645.

[18] Macdonald, M. E., and Cobb, S., this Journal, 1923–4, **4**, 228.

[19] Martin, J. P., *Lancet*, 1926, **1**, 760.

[20] Mosso, A., *Ueber der Kreislauf des Blutes im menschlichen Gehirn*, 1881.

[21] Muskens, L. J. J., *Epilepsy*, 1928.

[22] Nawratzki, E., and Arndt, M., *Berlin. klin. Woch.*, 1899, **36**, 662.

[23] Pollock, L. J., and Treadway, W. L., *Arch. Int. Med.*, 1913, **11**, 445.

[24] Redlich, E., and Pötzl, O., *Zeits. f. d. g. Neurol. u. Psychiat.*, 1910, **3**, 492.

[25] Robertson, Alexander, *Edin. Med. Jour.*, 1869–70, **15**, 513.

[26] Shephard, J. F., *Amer. Jour. Psychol.*, 1906, **17**, 522.

[27] id., *Amer. Jour. Physiol.*, 1909, **23**, xii.

[28] Sherrington, C. S., *Proc. Roy. Soc.*, 1909, **81B**, 249.

[29] Stenvers, H. W., *Deut. Zeits. f. Nervenheilk.*, 1925, **83**, 364.

[30] Stevenson, L., Christensen, B. E., and Wortis, B., *Proc. Assn. Research in Nervous and Mental Disease*, vol. VIII, 1929, p. 130.

[31] Todd, R. B., *Clinical Lectures on Paralysis*, 1854.

[32] Wilson, S. A. Kinnier, *Modern Problems in Neurology*, 1928.

APRAXIA IN CORPUS CALLOSUM LESIONS

REPORT OF THREE CASES

By

AARON BELL, New York

INTRODUCTION

In 1884 Bristowe [1] formulated a syndrome of tumours of the corpus callosum emphasizing the occurrence of profound mental disturbance, a tendency to double hemiparesis, difficulty in swallowing, loss of speech and want of control of the bladder, along with negative signs such as the absence of cranial nerve involvement and the mildness of signs due to increased intracranial pressure. Giese,[2] in 1892, and Ransom,[3] in 1895, reported one case each, Giese emphasizing the importance of the double hemiparesis, while Ransom, whose contribution included a review of the cases reported to that time, concluded that the diagnosis could not be made with certainty, but that the occurrence of profound mental disturbance with a double hemiplegia was suggestive of corpus callosum localization. Giese even went further and attempted localization in the different portions of the corpus callosum. In his conclusions, he makes the comment that when in addition to Bristowe's syndrome there are present signs of cerebellar involvement, then it is very likely that the lesion is in the splenium. Eight years later Zingerle [4] described a case of tumour of the corpus callosum in which the patient early in his illness showed psychic retardation, but on neurological examination manifested an inability to stand or walk even though the muscle power was good and there was no ataxia in the performance of non-equilibratory tests. This striking 'defect in coordination' which manifested itself in an inability to stand or walk in the absence of profound muscle paralyses or ataxia, Zingerle attributed to involvement of the corpus callosum and called it 'callosal ataxia' ('Balken-Ataxie'). In 1905, and again in 1907, Liepmann [5] made fundamental contributions to the study of the corpus callosum, pointing out that the corpus callosum was the pathway through which cerebral dominance may express itself. He described the occurrence of left-handed apraxia in right-handed individuals as diagnostic of corpus callosum lesions. Hartmann[6] confirmed Liepmann's findings, and Critchley,[7] in a comprehensive paper on the distribution of the anterior cerebral artery, again showed that when through a lesion of the callosal branch softening of the corpus callosum follows, left-handed apraxia is demonstrable clinically, providing, of course, the mental and physical state of the patient permits testing.

On the other hand, a good many investigators have attempted to interpret the mental picture encountered in corpus callosum lesions as specific for the corpus callosum. Bristowe had commented on the profound mental disturbance, which simulated that of general paralysis of the insane. In 1906 Raymond[8] reported two instances of corpus callosum tumour. Although he noted the absence of cranial nerve involvement and commented on the fact that one of his patients showed a disturbance in coordinating movements of his body when walking, Raymond felt that the mental picture was of more significance in localization. He regarded a disturbance in the association of ideas, a memory defect, particularly for recent events, an alteration in character, such as irritability and changing moods, and later a clouding of consciousness or dementia as a characteristic mental picture of corpus callosum lesions. Lippman [9] collected 50 cases from the literature, to which he added one of his own, and of the 51 cases only two were free of psychic disturbances. Lippmann also quotes Schuster's series of 33 cases, in 31 of which psychic disturbances were found. In 1914 Stern [10] in a detailed paper stated that the syndrome described by Raymond must be rare. His material did not substantiate the high incidence of pyschic disturbances as noted by Lippmann and Schuster. Stern felt that mental symptoms may be entirely absent, although frequently enough the mental symptoms appeared early, and were profound. Without regarding any specific psychic disturbances as diagnostic of the corpus callosum, Stern thought that when mental symptoms do occur they are likely to be of an amnesic nature resembling Korsakoff's syndrome and to consist also of defect in spontaneity. In their report of 14 cases of tumours of the corpus callosum, Ironside and Guttmacher [11] thought that mental changes such as apathy, drowsiness and defect in memory were often the first symptoms. Recently, Alpers and Grant [12] reported five cases of tumour of the corpus callosum formulating a syndrome which strongly emphasized the mental changes. According to their observation, difficulty in concentration and attention and defective comprehension are the characteristic mental changes in tumours of the corpus callosum. They write: 'A syndrome with the mental symptoms described and with hemiparesis and apraxia is almost characteristic of involvement of the corpus callosum. Less characteristic, but none the less indicative of callosal implication by tumour, is a syndrome consisting of pronounced mental symptoms with drowsiness and hemiparesis.' Alpers and Grant quote Mingazzini and Lévy-Valensi and Milani, all of whom felt that mental changes form the predominant symptomatology of corpus callosum disease.

It would seem, then, that in priority and accuracy of observation the syndrome described by Bristowe forms a sound nucleus for corpus callosum localization. Later investigations have either elaborated upon the mental changes encountered in the attempt to describe a specific psychic syndrome or have consisted of additions, the most significant being Liepmann's apraxia of the left hand. The difference of opinion in the literature as to the relative

significance of the mental changes and the physical findings may be accounted for either by the interval of time from the onset of the disease to the time when the patient comes under observation or more likely by the amount of neighbourhood invasion.

It is well known that growths of the corpus callosum invade the frontal lobes, and it may well be that the profundity of the mental alteration depends upon the extent of the spread into, and destruction of, the frontal lobes. It is also very likely that the severity of the pyramidal tract involvement is again an indication of the amount of pressure imposed upon, or of actual implication of, the internal capsules. If, then, as is likely, the paralysis and the personality changes are essentially neighbourhood signs, what remain of Bristowe's syndrome attributable directly to the corpus callosum are rather the negative signs, such as the lack of increased intracranial pressure and the rarity of cranial nerve involvement. On the other hand, the 'Balken-Ataxie' of Zingerle and the apraxia of Liepmann cannot be explained as neighbourhood signs. They appear to result from a disturbance in the corpus callosum itself.

Strictly speaking, the callosal ataxia of Zingerle is not an ataxia at all. Rather is it a disturbance in the execution of an idea in the presence of competent muscle power and a coordinating system which is intact when the patient is recumbent. As such it is an apraxia of gait. Although Liepmann's dictum that left-handed apraxia is a result of corpus callosum disease is correct, we would like to add that disease of the corpus callosum will produce disturbance in praxis which need not be confined to the arm, but may manifest itself in apraxia of the lower extremities or apraxia of the muscles of mastication or swallowing. In two of the three cases to be reported below, the localization of corpus callosum was made on the basis of an apraxia of gait; in the third, localization was made before the preagonal appearance of an apraxia of mastication and deglutition.

PERSONAL CASES

Case I.—Tumour involving the genu of the corpus callosum; history of 13 months' duration; apathy with drifting attention; mild bilateral pyramidal tract signs and apraxia of gait.

L.D., age 53, was admitted to the service of Dr. Foster Kennedy in August 1926. Her condition was alarming. She was in coma, respirations were stertorous and the pulse rapid and feeble. She was having a series of tonic fits suggestive of incomplete decerebrate rigidity. The head was twisted sharply to the right, the right arm was held rigidly flexed at the elbow, the left arm was extended and rigid, while the lower extremities were in extension spasm.

The history obtained from members of the family was that the patient had been quite well before September 1925. On Labour Day, 1925, she fell down a flight of stairs while under the influence of liquor. There was no loss of consciousness and no bleeding from the orifices. From that time she complained of headaches. In July 1926 she visited a public beach and went bathing in the nude. The family now noticed that her memory was very poor, that she lost interest in her home, and spent most of the time sleeping. When aroused, she complained of severe headache and on

several occasions the headache was accompanied by projectile vomiting. The past history was irrelevant.

Twenty-four hours after her admission the patient recovered from the initial coma and fits. Repeated examinations over a period of two months showed the following: She was apathetic but capable of normal emotional reactions. The two striking things about her mentality were an easy fatigability and an utter absence of any attempt to initiate activity. Her cooperation in the examination could not be kept longer than ten minutes at any one time. She was entirely unconcerned about her environment.

The pupils were equal, regular and reacted to light and upon accommodation. There were no ocular palsies and no nystagmus. The fundi were negative; the fields reconstructed after repeated examinations seemed to be intact. The motor and sensory fifth, including the corneal reflexes, were normal. There was a left central facial weakness, more apparent when the patient laughed. Hearing, phonation, palate, tongue and sternomastoid innervation were intact.

There was no frank paresis. Power of the grips and lower extremities was good. The patient executed movements slowly and deliberately. Finger-to-nose, heel-to-knee, and toe-to-object tests showed no disturbance in coordination. There was no apraxia in the hands.

The deep reflexes in the upper and lower extremities were grossly but equally overactive. The abdominal reflexes were not elicited. There was a bilateral Babinski sign.

Joint position sense was intact. Objects placed in the hands were readily recognized. There was no demonstrable change to pin-prick, touch and temperature.

In testing the gait, it was noted that the patient could not sustain herself. At times she would fall backwards, at other times she would collapse in a heap before the examiner. When supported and asked to take a few steps, the patient made no attempt to walk. The entire weight of her body was thrown upon the examiner and she seemed puzzled as to what was wanted of her.

The laboratory data showed an NPN of 40, a blood sugar of 130. The cerebrospinal fluid was clear, under normal pressure and had 40 lymphocytes per c.cm. The blood and spinal fluid Wassermann was negative.

Her behaviour during almost two months of hospitalization is best described as vegetative. She slept most of the day. She ate if and when she was fed. She was incontinent of fæces and urine and was content to lie in the evacuations until she was attended to. She was never heard to complain and never seen to speak to the patients about her. She died suddenly on the day she was to be operated upon.

The post-mortem examination was limited to the head. Inspection of the brain showed an ironing out of the right frontal convolutions. There was a slight bulging of the right frontal lobe. Upon separation of the two hemispheres, a bulging of the medial aspect of the right frontal region became apparent. Coronal sections of the brain, approximately 1 cm. apart, showed a soft cystic mass in the right frontal lobe extending from the second to, and including, the sixth section. The tumour had invaded and destroyed the genu and anterior portion of the corpus callosum. Microscopically, the tumour was a glioma.

Case II.—Granuloma compressing the genu of the corpus callosum: history of nine months' duration; choked discs, generalized weakness, mild pyramidal tract signs, apraxic gait and urinary incontinence. Suboccipital decompression with exitus.

C.P., coloured, single, 31 years of age, domestic, was admitted to the service of Dr. Foster Kennedy on October 25, 1932. The history was that since January 1932 she had been suffering from severe ' pressure ' headaches, localized to the vertex and radiating to the nape of the neck. The headaches were slightly relieved in the recumbent position. At no time were they associated with nausea or vomiting. In October of the same year she became aware of a tendency to drift to the left when walking. Gradual weakness of the left lower and upper extremity set in and at times the left arm would ' go dead.' Just before admission, she began to suffer from urinary incontinence, occurring both day and night, the urine escaping without her knowledge of any urge to urinate. At no time did she have vomiting, visual disturbances, uncinate phenomena, tinnitus or convulsions. She denied venereal infection.

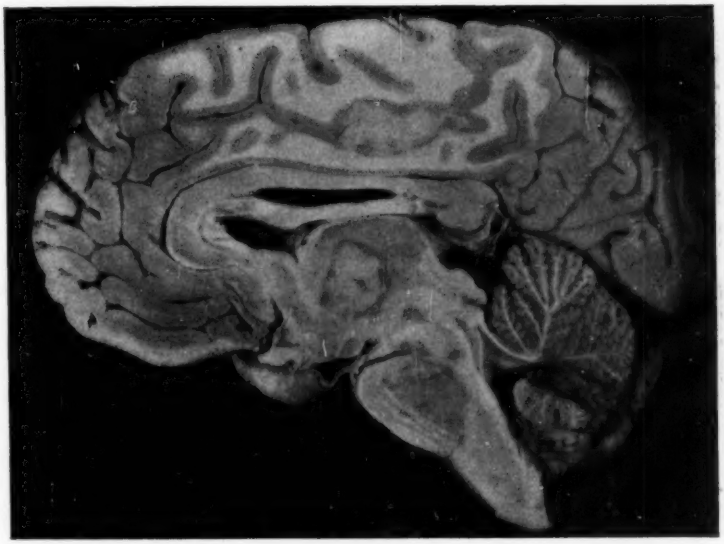


FIG. 1.—Sagittal section of brain in Case II, showing the two granulomatous nodules. The compression of the corpus callosum is not as evident in the photograph as it actually was in the specimen.

On examination the patient was well nourished. She cooperated well in the examination, though she seemed bewildered by the intricacies of the tests. There was some mental retardation, yet considering her endowment the latent period between a command and her response did not seem unduly prolonged. Spatial and temporal orientation was intact. She showed no delusional or hallucinatory formations. She was apprehensive about the nature of her illness.

The neurological status revealed a bilateral papillœdema with recent and old hæmorrhages. There was no anosmia. Visual fields showed general constriction. The pupils were equal, regular and reacted promptly to light and upon accommodation. There was weakness of the right internal rectus muscle upon convergence. There was no nystagmus. The muscles of mastication, sensation over the face, the corneal reflexes, the volitional and mimetic innervation of the facial muscles, hearing and the movements of the palate and tongue were normal. There was no aphasia.

There was a moderate amount of weakness in all the muscles, but no paralysis. The deep reflexes were equally overactive in the upper and lower extremities. The abdominal reflexes were diminished and the plantars were equivocal.

Non-equilibratory coordination tests were executed fairly well with the upper and lower extremities. There was no defect in the sensory status. There was no astereognosis and no apraxia in the upper extremities.

She could not stand without support. When with assistance she attempted to walk, the gait was bizarre. In walking, she would twist the body to one side, then raise the opposite leg much higher than was necessary or usual and bring it down as though she were uncertain as to where the floor was or how to make contact with the floor.

One week after admission ventriculography was performed. Following this procedure, nystagmus on lateral and upward gaze appeared, and in view of the fact that the ventriculogram showed a marked bilateral hydrocephalus without convincing localization of the third ventricle, a suboccipital exploration was performed, though up to the time of the ventriculography it had been postulated that she had a tumour of the corpus callosum. She died several days after the exploration.

Post-mortem examination was limited to the head. The gross appearance of the brain was that of an internal hydrocephalus, with flattening of the convolutions and distended ventricles. A sagittal section of the brain through the corpus callosum showed two granulomatous nodules, one nodule in the thalamus, the other in the gyrus cinguli immediately above the nodule in the thalamus. The two nodules had compressed the corpus callosum between them (fig. 1). Microscopically, the nodules were typical granulomas, probably luetic in origin.

COMMENT ON CASE I AND CASE II

Case I showed the characteristic picture described by Bristowe. Mental signs came early in the disease and the personality deterioration was profound. Signs of increased intracranial pressure were slight, the cranial nerve involvement was limited to a left central facial paresis, and there were bilateral pyramidal tract signs. Noteworthy in the mental picture was the lack of spontaneity and of all interest in her environment. She came under observation, vegetated for two months and died. One sees such personality alteration in frontal lobe tumours, and in this case there is every reason to believe that the profound mental change was due to extensive frontal lobe destruction. The one feature of this case which is not seen in frontal lobe tumours was the disturbance in locomotion.

By way of contrast with the first case, the second patient showed signs of increased intracranial pressure and no mental or emotional defects save a justifiable anxiety. It is worthy of note that in the second case the pathology was discrete, that there was no encroachment upon the frontal lobes and that there was very real compression of the corpus callosum. The unusual disturbance in locomotion permitted an approximately accurate localization of the pathological process.

In both patients locomotion was affected; in Case I it was impossible even with aid, and in Case II most bizarre with support and impossible

without aid. Both patients had forgotten how to walk even though muscle power was adequate, and there was no incoordination in executing tests in the recumbent position.

Reference to disturbance in station and locomotion in tumours of the corpus callosum is frequent in the literature. Bristowe's fourth patient 'could only walk with assistance and had a tendency to walk on her heels and fall over backwards.' Giese's patient had a tendency to veer to the right when walking. Zingerle's patient was unable to stand or walk. One of Raymond's patients walked as though intoxicated. One of Stern's patients fell slowly backward when attempting to stand. Lippmann's patient could not walk without support. Twelve of the 51 patients reviewed by Lippmann showed an unsteadiness in gait, and significant is his comment that in 12 the tumour was almost confined to the corpus callosum with very little neighbourhood invasion.

As has been indicated, Zingerle was the first to call attention to this disturbance in station and locomotion as a callosal ataxia. But since this disturbance in station and gait, which may vary from inability to walk without assistance to an absolute inability to walk even with aid, as in the first case reported here, is out of all proportion to the mildness of the motor disability and is present in the absence of any demonstrable incoordination when the patient is tested in the recumbent position, it becomes apparent that in callosal lesions one is not dealing with an ataxia at all. Rather it is more accurate to look upon this disturbance in gait as a loss of a specialized function : that is, that individuals with this disturbance do not know how to use their lower limbs for walking. As such, this disturbance in gait is an ideomotor apraxia, comparable to the apraxia in the left upper extremity which is at times demonstrable in lesions of the corpus callosum.

The two patients reported above, when tested in bed, showed good muscle power, recognized changes in the position of the joints, and executed the heel-to-knee, toe-to-object, and finger-to-nose tests without any difficulty, in spite of which they showed a gross disorder in station and locomotion. This is an apraxia of gait which results according to Liepmann from interruption in the interhemispherical conduction of impulses. It may be mentioned that an apraxia of the lower extremities could manifest itself only by such a disturbance in locomotion and gait as found in the patients reported.

Case III.—Degenerative lesion of the middle third of the corpus callosum ; history of eleven months' duration ; pseudobulbar syndrome with emotional explosiveness ; quadriplegia and bilateral pyramidal tract signs ; apraxic phenomena.

M.L., age 46, secretary, was admitted to the service of Dr. Foster Kennedy on January 2, 1933. In February 1932 the patient was in an automobile accident. There was no loss of consciousness and no bleeding from the orifices. In March of the same year she began to have difficulty with the left upper extremity. At first, objects which she held under the left arm would fall to the ground ; then weakness of the left arm appeared. Towards the end of March she had a left-sided convulsion without loss of consciousness. After a few days in bed she was able to continue at work. Her

employer now noticed that she had become unreliable and that her memory was not as good as it had been. In May she consulted physicians at Clifton Springs for the weakness of the left upper extremity. A small bone flap was turned down over the right parietal cortex, following which there was some recovery in the arm. Four days after returning home she had another left-sided convulsion. The weakness now involved the entire left side and she returned to Clifton Springs. In August a ventriculogram was done. Both the radiograms and the spinal fluid were negative at this time. In November of the same year, weakness of the right side started, first in the leg and then in the arm. Her speech became slurring; she could not control her evacuations; and she became subject to outbursts of tears and laughter. During the day she was very drowsy, though she could not sleep at night.

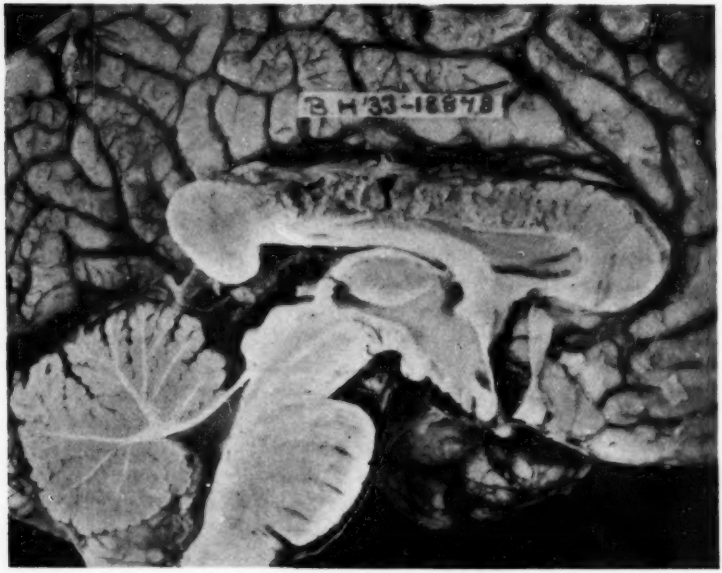


FIG. 2.—Sagittal section of brain in Case III, showing the degeneration of the middle two-thirds of the corpus callosum.

The past history was irrelevant, except that from the ages of 10 to 30 she had had attacks of migraine. The patient's mother, a sister and brother had similar attacks.

Examination upon admission showed a moderately obese middle-aged woman who was utterly helpless. She cooperated well in the examination. The fundi were negative and the visual fields seemed to be intact. The right pupil was larger than the left. Conjugate movement of the eyes to the right was not as good as to the left. There was a left central facial weakness. The tongue deviated to the left. Speech was muffled and indistinct, typically pseudobulbar. There was no aphasia.

There was a double hemiplegia with greater incidence on the left. She could do nothing for herself, the weakness being such that she had to be turned in bed and had to be fed. The deep reflexes were grossly overactive in the upper and lower extremities

and there was a bilateral Babinski sign, and bilateral Hoffmann sign. Praxis in the upper extremities could not be tested. There were no sensory changes demonstrable. She was incontinent of fæces and urine.

A lumbar puncture showed an initial pressure of 260 mm. of water. There was no block. The fluid was clear, had 4 lymphocytes to the c.cm., a total protein of 90 mgm., and the serology was negative.

She was a difficult patient for a ward. Periodically she became confused and disoriented. At such times she would be noisy and restless. Explosive outbursts of laughter alternated with sudden unpredictable outbursts of weeping. *A week after admission it was noted that the patient did not open her mouth or protrude her tongue, or deviate the eyes when asked to do so. She held food in the mouth for a long time before swallowing. Spontaneous movements of the eyes and jaws were, however, intact.* Her condition remained stationary and she died of intercurrent infection six weeks after admission.

Post-mortem examination of the brain showed the right frontal lobe to be smaller than the left, particularly in the neighbourhood of the longitudinal fissure about the motor region and in front of it. This region felt soft to the touch. There was, in addition, marked softening of the corpus callosum, particularly in the middle third. Sections of the softening showed a tremendous number of large compound granular corpuscles filled with fat. There were also a great many mononuclear cells resembling small and large lymphocytes. Many of the smaller cells contained fat. There was complete destruction of the myelin in the softened areas and no evidence of neuroglia proliferation.

COMMENT ON CASE III

This patient presented a picture of a progressive lesion of the brain with acute episodes resulting in a pseudobulbar syndrome. The double hemiplegia and the emotional lability were profound. Late in the disease the patient did not protrude the tongue, open the mouth or move the eyes when asked to do so. In this connexion it is interesting to point out that in 1909 Mills [13] reported a case of tumour of the frontal subcortex and callosum, where the man 'could not put out his tongue' when asked to do so, and that Bristowe's [1] third patient 'did not fully open his mouth or protrude his tongue when told to.' In the patient here reported, the failure to put out the tongue, open the mouth or deviate the eyes at a command appeared late in the disease, but at a time when the power of the jaw muscles and the muscles of the tongue was good. It is permissible to look upon these signs as apraxic phenomena resulting from a lesion in the corpus callosum.

CONCLUSION

The syndrome of the corpus callosum described by Bristowe is essentially a syndrome resulting from neighbouring involvement of the frontal subcortex. In all probability mental symptoms, when they do occur in callosal disease, follow the structural disturbance in the frontal lobes. However, the attempt to formulate a mental syndrome specific for the corpus callosum has not

been successful. This is not at all surprising, since the mental reaction of an individual to internal or external trauma is conditioned by the total personality equation of the individual. That being so, mental symptoms in organic disease of the brain depend less upon the location of the pathology than upon the personality 'make-up' of the individual.

Disturbances in praxis when not due to disease of the supramarginal gyrus are specific for corpus callosum pathology. The left-handed apraxia described by Liepmann is well known. In this communication other apraxic phenomena referable to lesions in the corpus callosum are described, such as apraxia of gait and apraxia of mastication and swallowing.

What in this paper has been called an apraxia of gait was described by Zingerle in 1900 as a 'Balken-Ataxie.' This apraxia of gait is an inability to use the lower extremities for walking in the presence of good muscle power and in the absence of any ataxia when the patient is tested in the recumbent position.

Three case histories are presented. In each a diagnosis of corpus callosum disease was made before exitus. The first two showed an apraxia of gait, the last an apraxia of the tongue and jaw muscles, in the course of an intracranial focal lesion. It is the purpose of this paper to emphasize the importance of apraxic phenomena in corpus callosum localization.

REFERENCES

[1] BRISTOWE, J. S., 'Cases of tumour of the corpus callosum,' *Brain*, 1884, **7**, 315.

[2] GIESE, 'Zur Casuistik der Balkentumoren,' *Arch. f. Psychiat.*, 1892, **23**, 961.

[3] RANSOM, W. B., 'On tumours of the corpus callosum with an account of a case,' *Brain*, 1895, **18**, 531.

[4] ZINGERLE, H., 'Zur Symptomatik der Geschwulste des Balkens,' *Jahrb. f. Psych.*, 1900, **19**, 367.

[5] LIEPMANN, H., and MASS, O., 'Fall von linksseitiger Agraphie und Apraxie bei rechtsseitiger Lähmung,' *Jour. f. Psychol. u. Neurol.*, 1907, **10**, 214.

[6] HARTMANN, F., 'Beiträge zur Apraxielehre,' *Monats. f. Psychiat. u. Neurol.*, 1907, **21**, 250.

[7] CRITCHLEY, M., 'The anterior cerebral artery,' etc., *Brain*, 1930, **53**, 120.

[8] RAYMOND, F., 'Tumeurs du corps calleux,' *L'Encéphale*, 1906, **6**, 533.

[9] LIPPMANN, A., 'Zur Symptomatologie und Pathologie der Balkentumoren,' *Arch. f. Psychiat.*, 1908, **43**, 1193.

[10] STERN, F., 'Die psychischen Störungen bei Hirntumoren,' *Arch. f. Psychiat.*, 1914, **54**, 565.

[11] IRONSIDE, R., and GUTTMACHER, M., 'The corpus callosum and its tumours,' *Brain*, 1929, **52**, 442.

[12] ALPERS, B. J., and GRANT, F. C., 'The clinical syndrome of the corpus callosum,' *Arch. of Neurol. and Psychiat.*, 1931, **25**, 67.

[13] MILLS, C. K., 'Tumours of the frontal subcortex and callosum,' *Jour. Nerv. Ment. Dis.*, 1909, **36**, 261.

CONGENITAL SYPHILIS IN MENTAL DEFECTIVE ADULTS

By

K. C. L. PADDLE, Caterham Mental Hospital

The relation of congenital syphilis to mental deficiency has for long been the subject of numerous investigations. In 1912 Mott [10] stated that 'if syphilis can produce arrest of development of the reproductive organs there is surely no reason why it should not lead to arrest of development of the most highly differentiated and specialized tissue of the body, viz., the cerebral cortex, or cause pathological variations in its structure and function.'

Since the introduction of the Wassermann reaction in 1906, an extensive literature dealing with the incidence of positive Wassermann reactions in mental deficiency has made its appearance, unfortunately with widely divergent results.

Table I

Showing Incidence of Positive Wassermann Reactions in the Blood-Serum of Mental Defectives

Authors	Date	Ages	Number of Cases	Positive Wassermann Reactions	Percentage
Raviart, Breton,[15] Petit, Gayer, etc. (France)	1908	All ages	246	76	30·8
Dean, H. R.[4] (Germany)	1909	do.	330	51	15·4
Thomsen, Boas, Hjort,[21] etc. (Germany)	1911	do.	2,061	31	1·5
Fraser and Watson [6] (England)	1913	1 to 17	194	91	46·9
Gordon, J. L.[7] (England)	1913	All ages	400	66	16·5
Roubinovitch, Baruk,[18] etc. (France)	1922	2½ to 17	48	15	31·0
Stewart, R. M.[20] (England)	1925	All ages	800	158	19·75
Dayton, N. A.[2] (U.S.A.)	1925	do.	9,183	497	5·4
Woodall, C. S.[22] (U.S.A.)	1928	do.	1,314	18	1·37
Bronfenbrenner, A. N.[1] (U.S.A.)	1930	do.	2,463	134	5·4

It will be seen that the results vary from 46·9 per cent. to as low as 1·37 per cent. It is probable that the relative sensitivity of the Wassermann reaction in different hands, and the method of interpretation, have had their influence. Kline [8] mentions the fact that the antigens employed in the majority of precipitation and complement-fixation tests for syphilis contain acetone-soluble adventitious substances 'which not only increase the sensi-

tivity of results, but also cause false positive reactions, especially when cholesterin in any amount is added.' There are also other factors which influence the incidence of positive Wassermann reactions, such as age, type and grade of defective, and even sex, as will be shown later.

PERSONAL RESEARCHES

During 1932 and 1933 it was decided to review the whole subject by a thorough investigation into the incidence of congenital syphilis amongst 2,000 * mental defectives of both sexes and all grades, inmates of Caterham Mental Hospital.

Bronfenbrenner [1] has expressed the opinion that 'statistics on the positive Wassermann reaction do not reflect adequately syphilogenic processes and, therefore, cannot be used by themselves for evaluation of syphilis as a factor in the ætiology of feeblemindedness.' Our enquiry has been based on the Wassermann reaction and Meinicke macro clarification reaction (M.K.R.) of the blood serum in all cases, as well as an examination of the cerebrospinal fluid in 1,927 cases, by the Wassermann reaction, cell estimation, globulin and Lange's colloidal gold tests. In addition, clinical examinations of all suspected cases were undertaken, and family histories obtained wherever possible. The tests on the blood and cerebrospinal fluid were carried out at the Central Pathological Laboratory of the London County Mental Hospitals by Dr. S. A. Mann, to whom I am indebted for his unfailing courtesy and kind cooperation in this work. The technique employed is fully described in a memorandum by Mann and Partner.[9] Briefly, for the Wassermann reaction, the figure following the first plus indicates the number of units of complement (1 unit equals approximately 0·01 c.c. of pure guineapig serum) absorbed by 1 c.c. blood-serum or cerebrospinal fluid. The plus following the figure indicates that a further absorption of complement would take place if the range of dilution of serum or cerebrospinal fluid were extended.

For the Meinicke macro clarification test (M.K.R.), four tubes are put up. A positive reaction is indicated by complete clarification of at least one tube.

The method of cell estimation used for the cerebrospinal fluid is only a rough one, 5 c.c. being centrifuged, filmed and stained, and a general expression of the number of cells present given in terms of one to four plus, one plus being equivalent to 15–20 cells per c.mm.; two plus, 20–30 cells; three plus, 30–40 cells; and four plus, over 40 cells. The results of the Pandy reaction for excess of globulin in the cerebrospinal fluid are given in terms of one to four plus, according to the density of the cloud produced, when two or three minims of fluid are dropped into a saturated solution of carbolic acid in a test tube at room temperature.

* Numbered 1—2000 for reference.

Among our 2,000 patients were 402 children of 16 years and under, all of whom had both blood and fluid examined. These formed a special group and were separately dealt with in a paper published in the *British Journal of Children's Diseases* of October, 1933 [Paddle [13]], many items of which I am quoting by kind permission of the Editor.

TOTAL NUMBER OF CONGENITAL SYPHILITICS

The remaining 1,598 adult cases are now reviewed. Of these, 1,492 were found to be serologically negative, and 106 gave various abnormal reactions in their blood-serum, cerebrospinal fluid or both. Excluding 10 cases of acquired syphilis, a full list of results is appended (Tables II—IV).

TABLE II

Congenital Syphilitic Male Defectives

Serial No.	Case No.	Age	Grade	Epilepsy	Cripple	Family History	Physical Signs	Blood-serum		Cerebrospinal Fluid				Remarks
								W.R.	M.K.R.	W.R.	Cells	Pandy	Lange	
1	87	41	Imb.L.	+	—	N.K.	±	+30+	++++	—	—	—	—	
2	138	32	F.M.	—	—	N.K.	±	+30+	++++	—	—	—	—	
3	150	18	Imb.M.	—	—	N.K.	+	+30+	++++	—	—	—	—	
4	464	19	F.M.	—	—	+	±	—	+++	—	—	—	—	
5	1,143	17	Imb.L.	—	—	N.K.	±	—	++	—	—	—	—	
6	1,205	25	Imb.M.	—	—	N.K.	+	+30+	++++	—	—	—	—	
7	1,232	23	Imb.L.	+	—	N.K.	+	+15	++++	—	—	—	—	
8	1,239	19	Imb.M.	—	—	N.K.	—	—	+++	—	—	—	—	
9	1,251	24	Imb.L.	—	+	N.K.	—	+30+	+++	—	—	—	—	
10	1,261	33	Imb.H.	+	—	N.K.	±	+10	++++	—	—	—	—	
11	1,265	20	Imb.L.	—	—	N.K.	+	+30+	++++	—	—	—	—	Choroiditis
12	1,269	17	Imb.L.	—	—	±	±	—	+++	—	—	—	—	
13	1,278	20	F.M.	—	+	N.K.	±	—	+++	—	—	—	—	
14	1,319	18	Imb.H.	—	+	N.K.	+	+15	+++	—	—	—	—	
15	1,346	24	Imb.M.	—	—	N.K.	±	—	++	—	—	—	—	
16	1,360	21	Imb.L.	—	—	+	±	—	+++	—	—	—	—	
17	1,376	23	Imb.M.	+	+	N.K.	+	+30+	++++	—	—	—	—	
18	1,382	28	Imb.L.	+	—	N.K.	±	—	++	—	—	—	—	
19	1,415	31	Imb.L.	+	—	N.K.	±	+10	++	—	—	—	—	
20	1,424	23	F.M.	+	—	N.K.	±	—	+++	—	—	—	—	Choroiditis
21	1,441	23	Imb.M.	+	—	—	—	—	++	—	—	—	—	
22	1,504	35	Imb.M.	—	—	N.K.	+	—	++	—	—	—	—	
23	1,571	18	Imb.L.	—	—	+	+	+30+	++++	—	—	—	—	Mongolian
24	1,581	38	F.M.	—	—	N.K.	±	—	+++	—	—	—	—	
25	1,618	23	Imb.L.	+	—	+	±	+30+	++++	—	—	—	—	
26	1,672	30	Imb.H.	—	—	+	+	—	+	—	—	—	—	
27	1,714	54	Imb.M.	—	—	N.K.	+	—	++	—	—	—	—	
28	1,768	29	F.M.	—	—	N.K.	±	+30+	++++	—	—	—	—	
29	1,813	42	Imb.M.	—	—	N.K.	±	—	+++	—	—	—	—	
30	1,827	60	Imb.M.	—	—	N.K.	±	+30+	+++	—	—	—	—	
31	1,845	57	Imb.M.	+	+	N.K.	+	—	+++	—	—	—	—	
32	1,856	64	Imb.M.	—	—	N.K.	+	L	++	—	—	—	—	
33	1,979	23	F.M.	—	—	N.K.	+	+30	+++	N.D.	N.D.	N.D.	N.D.	
34	64	21	Imb.L.	+	+	+	+	—	—	+6+	+	++	5555554321	Juvenile G. P.
35	497	62	Imb.M.	—	—	N.K.	—	+30	++++	—	+	+	—	
36	1,151	17	Imb.L.	—	—	+	±	—	—	—	++	—	—	
37	1,250	20	Imb.L.	—	+	N.K.	+	+30+	++++	+15+	—	+	4433210000	Choroiditis
38	1,339	26	Imb.M.	+	—	N.K.	+	—	—	—	+	—	—	
39	1,612	25	Imb.L.	—	—	N.K.	—	—	—	—	—	—	—	W.R.++ in 1929

Table IV represents a group of 21 cases, many of which had abnormal fluids, others gave weak positive M.K.R.'s, and in one case a doubtful positive

TABLE III

Congenital Syphilitic Female Defectives

Serial No.	Case No.	Age	Grade	Epilepsy	Cripple	Family History	Physical Signs	Blood-serum		Cerebrospinal Fluid				Remarks
								W.R.	M.K.R.	W.R.	Cells	Pandy	Lange	
1	19	44	Imb.L.	—	—	N.K.	+	+30+	+++	—	—	—	—	
2	35	47	Imb.L.	+	—	+	+	+30+	++++	—	—	—	—	Deaf mute Choroiditis
3	43	55	Imb.L.	+	—	N.K.	+	+30+	++++	—	—	—	—	
4	216	19	Imb.L.	+	—	+	±	+30+	—	—	—	—	—	
5	360	27	Imb.M.	+	—	+	+	+30+	—	—	—	—	—	Choroiditis
6	474	19	Imb.L.	+	+	—	+	—	+	—	—	—	—	
7	573	58	Imb.L.	—	—	N.K.	—	+30+	+	—	—	—	—	Microcephalic
8	608	30	F.M.	+	—	+	—	+30+	++++	—	—	—	—	Choroiditis
9	611	73	Imb.H.	—	—	N.K.	+	+30+	++++	—	—	—	—	
10	610	63	Imb.H.	—	—	N.K.	+	+30+	++++	—	—	—	—	Choroiditis
11	614	41	Imb.L.	—	—	N.K.	±	+30+	++++	—	—	—	—	
12	745	34	Imb.L.	—	—	N.K.	±	—	++++	—	—	—	—	
13	756	30	F.M.	+	—	+	±	+30+	++++	—	—	—	—	Hydroce-[phalic
14	765	17	Imb.L.	+	—	N.K.	+	—	++++	—	—	—	—	Blind
15	774	19	F.M.	—	—	N.K.	+	—	+++	—	—	—	—	Choroiditis
16	783	29	Imb.M.	+	+	+	+	+30+	++++	—	—	—	—	
17	791	18	Imb.L.	—	—	N.K.	+	+30+	++++	—	—	—	—	
18	884	45	Imb.L.	—	—	N.K.	±	+15	+++	—	—	—	—	
19	896	44	Imb.L.	—	—	N.K.	+	+30+	++++	—	—	—	—	
20	917	26	Imb.L.	—	—	N.K.	±	—	+	—	—	—	—	
21	955	29	Imb.H.	—	—	N.K.	+	+30+	++++	—	—	—	—	Blind
22	970	20	Idiot	+	—	N.K.	±	—	+++	—	—	—	—	
23	994	28	Imb.H.	+	—	+	+	+30+	++++	—	—	—	—	
24	1,050	36	Imb.M.	—	—	N.K.	±	+30+	++++	—	—	—	—	
25	1,923	58	Imb.M.	—	—	N.K.	+	—	+	—	—	—	—	
26	1,994	34	Imb.M.	—	—	N.K.	+	++	N.D.	N.D.	N.D.	N.D.	N.D.	Died of Banti's disease
27	41	49	Imb.H.	—	—	N.K.	+	—	—	+	—	—	2222110000	
28	116	17	Idiot	+	+	N.K.	±	+30+	++++	+30+	++	+	4433210000	
29	135	29	F.M.	—	—	N.K.	±	+30	—	+3+	+	—	—	
30	248	45	Imb.L.	—	—	N.K.	+	+30	++++	+3+	+	+	2221100000	
31	569	52	Imb.M.	—	—	N.K.	+	+30	+	—	—	+	0001221000	
32	600	65	Imb.M.	—	—	N.K.	+	+30+	+++	—	+	+	—	Choroiditis
33	613	45	Imb.H.	—	—	N.K.	+	+30+	++++	+30+	++	++	5555443210	
34	1,351	30	Imb.H.	—	—	+	+	—	—	+6	—	—	—	Choroiditis
35	82	17	Imb.L.	—	—	—	+	—	—	—	—	+	2221110000	Choroiditis Deaf mute
36	350	48	Imb.M.	+	—	—	+	—	—	—	—	+	—	
37	801	23	Imb.L.	—	—	N.K.	+	—	—	—	+	+	5554321000	Mongolian
38	619	46	Imb.M.	—	+	N.K.	+	—	—	—	—	—	—	
39	620	21	Idiot	+	—	N.K.	+	—	—	—	—	—	—	

Wassermann reaction of the blood-serum. It is possible that some of these cases may be of syphilitic origin, but on account of lack of confirmatory evidence they have been classed as non-syphilitic. This leaves 75 cases of congenital syphilis, to which must be added three serologically negative cases, two (nos. 619 and 620) with undoubted clinical signs of congenital syphilis, and one (no. 1,612) with previous positive Wassermann reaction of the blood serum, making 78 in all.

INCIDENCE OF CONGENITAL SYPHILIS

Of the 78 cases of congenital syphilis the Wassermann reaction was positive in 43, or 55·1 per cent., and the M.K.R. was positive in 63, or 80·8 per cent. The latter test is, therefore, the more sensitive one. In the special

TABLE IV

Abnormal Serological Reactions in Non-syphilitic Defectives

Serial No.	Case No.	Sex	Age	Grade	Epilepsy	Cripple	Family History	Physical Signs	Blood-serum		Cerebrospinal Fluid				Remarks
									W.R.	M.K.R.	W.R.	Cells	Pandy	Lange	
1	31	Male	26	Imb.L.	—	—	N.K.	—	±	—	—	—	—	—	
2	75	,,	43	Imb.M.	—	—	N.K.	—	—	—	—	+	+	0012321000	
3	137	,,	44	Imb.M.	—	—	N.K.	—	—	—	—	+	+	—	
4	169	,,	26	Imb.H.	—	—	N.K.	—	—	—	—	+	—	—	
5	504	,,	19	Imb.M.	—	—	N.K.	±	—	+	—	—	—	—	
6	1,168	,,	20	Imb.L.	+	+	N.K.	—	—	—	—	—	+	—	Quadriplegic
7	1,328	,,	28	Imb.L.	+	—	N.K.	—	—	—	—	—	+	1122211000	
8	1,334	,,	23	Imb.L.	+	—	N.K.	—	—	—	—	+	+	—	
9	1,358	,,	29	Imb.L.	+	+	N.K.	±	—	—	—	—	+	—	Diplegic
10	1,397	,,	39	Imb.M.	—	—	N.K.	±	—	—	—	—	—	—	
11	1,476	,,	26	Imb M.	—	—	N.K.	±	—	—	—	—	++	0000112321	
12	1,519	,,	20	Imb.H.	+	—	N.K.	±	—	—	—	+	+	—	
13	1,525	,,	54	Imb.L.	—	—	N.K.	—	—	+	—	—	—	—	
14	1,584	,,	35	Imb.M.	—	—	N.K.	—	—	—	—	++	—	—	
15	1,841	,,	70	F.M.	—	—	N.K.	±	—	+	—	—	—	—	
16	219	Female	36	Imb.L.	—	—	N.K.	—	—	—	—	—	+	—	
17	233	,,	17	Imb.L.	—	—	N.K.	—	—	—	—	++	—	—	
18	697	,,	17	Idiot	+	—	—	—	—	—	—	—	++++	0001122334	Epiloia
19	699	,,	40	Imb.L.	—	—	N.K.	—	—	—	—	—	+++	0001122321	
20	1,915	,,	55	Imb.H.	—	—	N.K.	—	—	+	—	—	—	—	
21	1,922	,,	70	Imb.H.	—	—	N.K.	±	—	+	—	—	—	—	

Idiot = Defective with an intelligence quotient of below 25.
Imb.L. = Imbecile of low grade with an intelligence quotient of 25–33.
Imb.M. = ,, medium grade with an intelligence quotient of 34–42.
Imb.H. = ,, high grade with an intelligence quotient of 43–50.
F.M. = Feebleminded with an intelligence quotient above 50.
Family History + = Definite evidence of syphilis in either or both parents.
,, ,, ± = Doubtful evidence.
Physical Signs + = Definite signs of congenital syphilis.
,, ,, ± = Doubtful signs of congenital syphilis.
N.K. = Not known.
N.D. = Not done.

group of 402 children the reverse was the case. Of the total 1,598 cases, excluding acquired syphilis, the incidence of positive Wassermann reactions works out at 2·7 per cent., that of positive M.K.R.'s at 3·9 per cent., and the total incidence of congenital syphilis at 4·9 per cent.

The incidence of positive Wassermann reactions in the group of 402 children was 6·7 per cent., and the total incidence of congenital syphilis 9·2 per cent. The lower rate in adults reflects the tendency for the Wassermann and parallel tests to fade with advancing age. One must, therefore, suspect that there are many adult congenital syphilitics giving negative serological reactions. The task of identifying such cases is made extremely difficult by the lack of clinical signs, and by increased difficulty in getting personal and family histories.

RELATIVE INCIDENCE OF CERTAIN GRADES AND DEFECTS

An analysis of the various grades of mental defect is given in Table V.

TABLE V

Relative Incidence of Grades

	Free from congenital syphilis		Congenital syphilitics		Percentage incidence of congenital syphilis in total number of cases
	Number	Per cent.	Number	Per cent.	
Idiot	135	8·9	3	3·9	2·2
Imbecile (low grade) . .	560	36·8	31	39·7	5·2
,, (medium grade) .	392	25·8	23	29·4	5·5
,, (high grade) . .	151	9·9	10	12·9	6·2
Feebleminded . . .	282	18·6	11	14·1	3·4
Totals . . .	1,520	100·0	78	100·0	

The imbecile group has the highest incidence of congenital syphilis, the idiot group the lowest, the feebleminded group occupying a middle position. It may be thought that factors such as epilepsy and paralysis would affect the results ; that this is not so is shown by the removal of such cases. There were 1,025 defectives composed of 56 idiots, 752 imbeciles and 217 feebleminded, all free from paralysis and epilepsy. Among the 56 idiots there were no cases of congenital syphilis, the imbeciles had 40 or an incidence of 5·3 per cent., and the feebleminded seven or 2·7 per cent., the relative incidence thus remaining unaltered. It is of interest to note that the same state of affairs prevailed amongst the group of 402 children, though the numbers were too few to draw any conclusions.

Woodall [22] found precisely the same relation to exist amongst the three grades. After excluding cases of hydrocephalus, microcephalus, paralysis, cretinism, and mongolism, he found the following :—

	Number of congenital syphilitics	Total number	Per cent.
Moron (feebleminded) . .	35	537	6·5
Imbecile	31	347	8·9
Idiot	12	216	5·6

Bronfenbrenner [1] and Dayton [3] have shown that the incidence of positive Wassermann reactions tends to be lower in the lower grades of mental defect. The former thinks that this is due to a ' degenerative phase of syphilis ' affecting idiots to a greater extent than imbeciles or feebleminded, such cases giving a negative Wassermann reaction, and therefore tending to lower the positive incidence in the lower grades of mental defect. This conception has been termed the ' intelligence quotient (I.Q.) gradient of the Wassermann reaction in the mental defective,' meaning that the higher the I.Q. the greater the incidence of positive Wassermann reactions, when large numbers of mental defectives of approximately the same age are examined.

It would seem probable that one of the chief factors influencing the rate of congenital syphilis among the grades in both our group of children and adults is the I.Q. gradient, and that there is a drop in this factor just above the imbecile level.

Congenital syphilis is known occasionally to cause paralysis and epilepsy. A higher rate of congenital syphilis is, therefore, to be expected in the cripple-epileptic class of mental defect. This is shown to be the case in Table VI, the uncomplicated group having the lowest incidence.

TABLE VI

Relative Incidence of Certain Defects

	Free from congenital syphilis		Congenital syphilitics		Percentage incidence of congenital syphilis in total number of cases
	Number	Per cent.	Number	Per cent.	
Epileptics (non-cripple) .	347	22·8	20	25·6	5·4
Cripple (non-epileptic) . .	76	5·0	5	6·4	6·2
Cripple and epileptic . .	72	4·7	6	7·7	7·7
Uncomplicated * . . .	1,025	67·5	47	60·3	4·4
Totals . . .	1,520	100·0	78	100·0	

* Not cripple or epileptic.

The incidence of congenital syphilis in certain mental defective types and diseases associated with mental deficiency is given in Table VII.

TABLE VII

Relation of Congenital Syphilis to Certain Physical Types and Diseases Associated with Mental Deficiency

	Children	Adults	Total	Congenital syphilitics	Per cent.
Mongolism	29	24	53	2	3·8
Deaf mutism . . .	8	6	14	2	14·3
Hydrocephalus . . .	7	10	17	3	17·6
Microcephalus . . .	12	10	22	2	9·1
Banti's disease . . .	0	1	1	1	100·0
Diabetes mellitus . .	0	4	4	1	25·0
Epiloia	4	12	16	Nil.	Nil.
Cretinism	2	5	7	—	—
Achondroplasia . . .	1	0	1	—	—
Fröhlich's syndrome . .	1	1	2	—	—
Choreoathetosis . . .	3	4	7	—	—
Post-encephalitis lethargica .	4	6	10	—	—
Meningeal angioma . .	0	1	1	—	—
Cerebromacular degeneration.	2	0	2	—	—
Pseudohypertrophic muscular paralysis	2	0	2	—	—
Schilder's encephalitis . .	1	0	1	—	—

The numbers are rather small, but the low incidence (3·8 per cent.) of congenital syphilis among mongols should be noted, as it has been suggested that there is a direct causal relationship between the two conditions. For example, Stevens [19] in 1916 found the Wassermann reaction positive in the blood-serum of six out of 18 mongolian idiots, the Wassermann reaction positive in the cerebrospinal fluid in two out of 18, and the gold chloride reaction present in 100 per cent. of the cases. He was of opinion that the serological tests demonstrated beyond question that the condition of mongolism was a result of syphilis, though he thought it probable that the latter acted primarily on some of the endocrine glands, possibly the pituitary body. Riddel and Stewart [16] have since confirmed Stevens' findings with regard to the property of the spinal fluid of mongols to precipitate solutions of colloidal gold in the luetic zone. They examined the fluid of 55 mongols and all except one gave a luetic curve. The Wassermann reaction was, however, negative in all the fluids, and the cells and globulin all normal; only one out of 55 gave a positive Wassermann reaction in the blood-serum. There were 53 mongol defectives (children included) in our series, all of whom had both blood and cerebrospinal fluid examined. The Wassermann reaction was positive in only one case in the blood-serum, and negative in all the fluids; in 52 cases the fluid failed to give any sort of curve with the colloidal gold test (the exception being no. 801), and there was no increase of globulin or cells in these specimens. It therefore appears that syphilis had little to do with the causation of mongolism in our 53 cases.

Banti's disease must be rare amongst mental defectives. Its association with syphilis has been noted by various authors, including Osler.[12] One such case occurred in our series (no. 1,994) of congenital syphilis, which went to a fatal termination, being fully reported upon [Paddle [14]].

Rolleston [17] in 1921 referred to the importance of endocrine infections in congenital syphilis. He thought 'it would be remarkable if the ductless glands escaped syphilization.' There was one congenital syphilitic in four cases of diabetes mellitus who after treatment with neoarsenobensol was able to dispense with daily injections of insulin. Table VII also shows that such conditions as cretinism, choreoathetosis and post-encephalitis lethargica appear to be unconnected with congenital syphilis.

A complete physical examination, including retinoscopy, was made of all the 78 cases of congenital syphilis, the results being given in Table VIII. One notes the comparative rarity of Hutchinson's and Moon's teeth. 52·6 per cent. of cases had other dental deformities such as described by Fournier,[5] but these defects are not considered by themselves to be diagnostic of congenital syphilis, as they are often met with in mental defectives apart from syphilis.

Bony lesions are fairly common in congenital syphilis. It is not, therefore, surprising to find that 47·4 per cent. of cases showed abnormal carrying

TABLE VIII

Signs Associated with Congenital Syphilis in 78 Cases

	No.	Per cent.		No.	Per cent.
Infantilism	7	8·8	Alopecia	4	5·1
Stunted growth	12	15·4	Pallor	12	15·4
Quadriplegia	1	1·3	Dactylitis	5	6·4
Diplegia	1	1·3	Onychia	5	6·4
Monoplegia	2	2·6	Scars of old ulcers	18	23·6
Hemiplegia	6	7·7	Nodes on bones	12	15·4
Strabismus (internal)	3	3·8	Chronic periostitis of tibia	10	12·8
,, (external)	1	1·3	,, ,, of femur	1	1·3
Iridoplegia	18	23·1	Curved tibia	4	5·1
Cataract	2	2·6	Lax ligaments of joints	3	3·8
Iritis	12	15·4	Coxa vara	1	1·3
Interstitial keratitis	18	23·6	Abnormal carrying angle—		
Choroiditis (disseminated)	10	12·8	At elbows	37	47·4
,, (diffuse)	1	1·3	At wrist	3	3·8
Atrophied eyes	2	2·6	Scaphoid scapula	1	1·3
Facies syphilitica	10	12·8	Absent ziphoid appendix	4	5·1
Skull asymmetrical	9	11·5	General glandular enlargement	2	2·6
Sunken bridge of nose	4	5·1	Splenic enlargement	2	2·6
Frontal bossing	27	34·6	Liver enlargement	2	2·6
Rhagades	11	14·1	Thyroid enlargement (symmetrical)	3	3·8
Prominent lips	27	34·6	Cirrhosis of liver	1	1·3
Scarring of palate	4	5·1	Birth palsy	1	1·3
Teeth : Hutchinson's	4	5·1	Unilateral synovitis of wrist and ankle	1	1·3
,, Moon's	2	2·6	Mitral stenosis	4	5·1
,, other defects	41	52·6	Undescended testes	3	7·6 (of 39)
Fissured tongue	13	16·7			
Cleft palate	1	1·3			
Eighth nerve deafness	16	20·5			
Bilateral dacryocystitis	6	7·7			

angles at the elbows. This consisted of a marked outward deviation of the forearm with limited extension in a number of cases.*

In the group of 25 congenital syphilitic boys between the ages of seven and 16, 15 or 60 per cent. had undescended testes; in the group of 39 male adults, only three or 7·6 per cent. had undescended testes, showing that this condition is really in the large majority of cases one of retarded descent, and not one of permanent arrest in the inguinal canal.

THE CEREBROSPINAL FLUID

Of 1,598 cases, 1,525 had their cerebrospinal fluid examined for increase in cells and globulin and the Wassermann reaction and Lange's colloidal gold test applied. The results are included in Tables II to IV.

Of these 1,525 cases (excluding 10 of acquired syphilis, four of which had abnormal fluids) the fluid was abnormal in 30, or 2 per cent. This compares with 5·2 per cent. in our previous group of 402 children, and 41 per

* I am indebted to Dr. R. M. Stewart for having indicated this sign to me.

cent. of 48 'mentally retarded children' reported by Roubinovitch [18] and his co-workers.

Of the 78 adult cases of congenital syphilis the fluid was examined in 76, and of these in 16, or 21 per cent., it was found to be abnormal. In the group of 37 congenital syphilitic children 12, or 32·4 per cent., had abnormal fluids. Among the 78 congenital syphilitics there was only one case of juvenile general paralysis; his fluid gave a positive Wassermann reaction, a paretic type of curve with the colloidal gold test, and an increase in cells and globulin. Eight other cases gave paretic types of curve, some weak, but none of these cases could on clinical grounds be regarded as juvenile general paralysis.

As already mentioned, the cerebrospinal fluid of 52 out of 53 mongols failed to give any type of curve with the colloidal gold reaction, the exception being a female mongolian imbecile considered on clinical grounds to be a case of congenital syphilis, but with negative Wassermann reaction and Meinicke macro clarification reaction of the blood-serum; her fluid also gave a negative Wassermann reaction, but there was an increase of cells and globulin and a colloidal gold curve of the paretic type. Of the 21 cases of doubtful origin (Table IV) 14 had abnormal fluids, mostly increases of globulin, six showed increase of cells and five gave colloidal gold reactions, mostly in the middle zone. It is difficult to say what these abnormal fluids represent, as no confirmatory evidence of syphilis was available, and no clinical signs to account for the findings. It is interesting to note that case no. 697, an epiloiac female idiot, has since died, and postmortem a tumour was found beneath the genu of the corpus callosum, bulging into both lateral ventricles, and obliterating by pressure the foramina of Munro, with consequent internal hydrocephalus. This would account for the large increase in protein and the type of colloidal gold curve found in the cerebrospinal fluid when she was alive. Choroiditis appears to be more common in congenital syphilitics with abnormal fluids than in those with normal fluids, the incidence in the former class being 25 per cent. and in the latter 11·7 per cent.

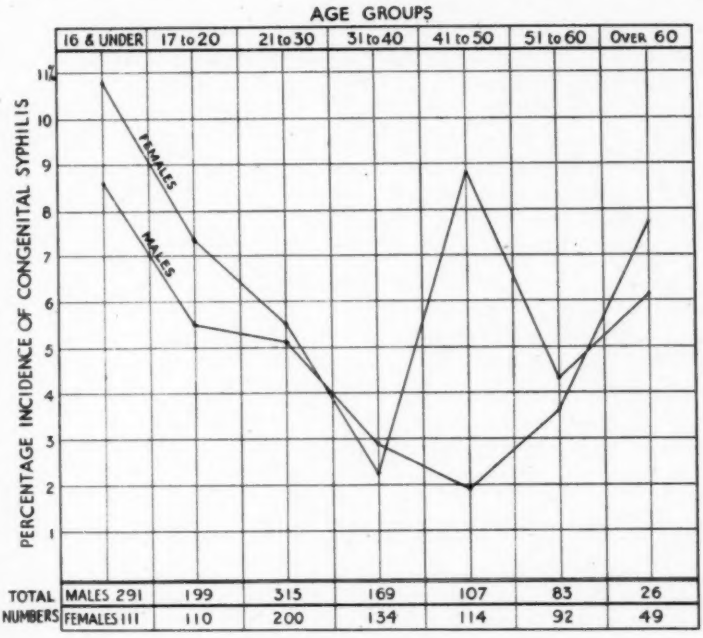
THE INFLUENCE OF AGE AND SEX

There were 899 males and 699 females, the incidence of congenital syphilis among them being 4·3 and 5·6 per cent. respectively.

If the males and females are separated into age groups (the 402 children being included) and the incidence of congenital syphilis plotted out in the form of a graph, it will be seen that the curves followed by the two sexes are unlike.

The curve for females starts higher than that of the males and is at its lowest between the ages of 31 to 40. It then rises sharply and is once more higher between the ages of 41 to 50, then falls, to rise again after the age of 60. The male curve, starting lower, falls more gradually, being lowest

between the ages of 41 to 50, then slowly rises, being higher again after 60. The initial fall in incidence is mainly due to the fading of the Wassermann and Meinicke reactions with advancing age. The higher incidence among females between the ages of 41 to 50 would suggest the influence of the menopause on these serological tests. Such a view has already been put forward by Nicole [11] when dealing with acquired syphilis in the insane. He examined 640 females and 989 males, inmates of Winwick Mental Hospital, and came to the conclusion that ' the menopause does affect the serological



reactions of females,' and ' many of the females examined before 50 give negative reactions, not because they are not syphilitic, but merely because they have not yet reached the menopause.'

SUMMARY

Fifteen hundred and ninety-eight adult mental defectives of all grades and both sexes had the Wassermann reaction and Meinicke macro clarification reaction done on the blood-serum. Fifteen hundred and twenty-five of these cases had their cerebrospinal fluid examined by the Wassermann reaction, Pandy's test, Lange's colloidal gold test, and for increase of cells. One hundred and six cases gave various abnormal reactions in the blood or cerebrospinal fluid, and 1,492 were serologically negative. Of the 106, 75

were considered to be congenital syphilitics, and of the 1,492 serologically negative cases, three were deemed on clinical and other grounds to be congenitally syphilitic—giving a total of 78 cases of hereditary syphilis, or an incidence of 4·9 per cent. The M.K.R. of the blood-serum in adults was more sensitive than the Wassermann reaction.

The incidence of congenital syphilis was higher among imbeciles than idiots or feebleminded, and higher in cripple-epileptics than in uncomplicated cases. There was no evidence to show that mongolism was caused by syphilis. Conditions such as cretinism, choreoathetosis and post-encephalitis lethargica were unconnected with congenital syphilis.

Of 1,525 cases, excluding 10 cases of acquired syphilis with four abnormal fluids, the cerebrospinal fluid was abnormal in 30, or 2 per cent.

In 76 cases of congenital syphilis the fluid was abnormal in 16, or 21 per cent.

The incidence of congenital syphilis was found to fall with advancing age but differed in the sexes. In females it was highest in children and lowest between 31 to 40, with a sharp rise between 41 to 50, suggesting a menopausal influence on the serological reactions used. At this age the incidence among the males was at its lowest, but there was a subsequent rise.

My thanks are due to Dr. T. Lindsay, Medical Superintendent of Caterham Mental Hospital, Dr. F. L. Golla, Director of the Central Pathological Laboratory of the London County Mental Hospitals, and Dr. R. M. Stewart, Medical Superintendent of Leavesden Mental Hospital, as well as to my many colleagues, for their helpful advice in this work. I am indebted to Dr. Lindsay for kind permission to publish details of these cases.

REFERENCES

[1] Bronfenbrenner, A. N., 'The Wassermann test in the feebleminded,' *Psychiat. Quarterly*, 1931, **5**, 45.

[2] Dayton, N. A., 'Congenital syphilis as a cause of mental deficiency,' *Boston Med. Surg. Jour.*, 1925, **193**, 668.

[3] Dayton, N. A., 'Degree of mental deficiency resulting from congenital syphilis,' *Jour. Amer. Med. Assoc.*, 1926, **87**, 907.

[4] Dean, H. R., 'Idiocy and congenital syphilis,' *Brit. Jour. Child. Dis.*, 1912, **9**, 385.

[5] Fournier, E., *Stygmates dystrophiques de l'hérédo-syphilis*, 1890, 70.

[6] Fraser, K., and Watson, H. F., 'The rôle of syphilis in mental deficiency and epilepsy,' *Jour. Ment. Sci.*, 1913, **59**, 640.

[7] Gordon, J. L., 'The incidence of inherited syphilis in congenital mental deficiency,' *Lancet*, 1913, **2**, 861.

[8] Kline, B. S., *Microscopic Slide Precipitation Tests for the Diagnosis and Exclusion of Syphilis*, 1932, 8.

[9] Mann, S. A., and Partner, F., *Memorandum on the Wassermann Reaction in Mental Hospitals' Practice*, 1931.

[10] Mott, Sir F., *Proc. Roy. Soc. Med.*, 1912, **5**, 80.

[11] Nicole, J. E., 'The age-incidence of seropositive syphilis in females,' *Brit. Med. Jour.*, 1932, **1**, 749.

[12] Osler and McCrae, *The Principles and Practice of Medicine*, 883.

[13] Paddle, K. C. L., 'Congenital syphilis in low-grade mentally defective children,' *Brit. Jour. Child. Dis.*, 1933, **30**, 249.

[14] Paddle, K. C. L., 'Atrophic coarse cirrhosis of the liver in a congenital syphilitic,' *Brit. Med. Jour.*, 1932, **1**.

[15] Raviart, G., Breton, M., Petit, G., Gayet and Cannac, 'Réaction de Wassermann et aliénation mentale,' *Rev. de méd.*, 1908, **28**, 840.

[16] Riddell, D. O., and Stewart, R. M., 'Syphilis as an ætiological factor in Mongolian idiocy,' *Jour. Neurol. and Psychopath.*, 1923, **4**, 221.

[17] Rolleston, Sir H., *Proc. Roy. Soc. Med.*, 1921, **14**, 46.

[18] Roubinovitch, J., Baruk and Barıety, 'Le liquide céphalorachidien dans la débilité mentale infantile simple,' *L'Encéphale*, 1922, **17**, 518.

[19] Stevens, H. C., 'Mongolian idiocy and syphilis,' *Jour. Amer. Med. Assoc.*, 1916, **66**, 1373.

[20] Stewart, R. M., 'The incidence of congenital syphilis in mental deficiency,' *Metropolitan Asylums Board Annual Report*, 1925–6, 213.

[21] Thomson, O., Boas, H., Hjort, R., and Leschly, W., *Berl. klin. Woch.*, 1911, **48**, 891.

[22] Woodall, C. S., 'The incidence of congenital syphilis in an institution for the feebleminded,' *Amer. Jour. Psychiat.*, 1929–30, **9**, 1065.

AN UNUSUAL TYPE OF CORTICAL GLIOSIS

By

R. M. STEWART

Leavesden

The peculiar type of cerebral gliosis which is the subject of the present communication was discovered postmortem in a case of muscular dystrophy, and is of special interest in its possible relationship to tuberous sclerosis, which may perhaps be regarded as the prototype of those developmental diseases in which circumscribed overgrowth of neuroglia is the outstanding feature. In addition to the pathological interest of the case, the terminal phase of the patient's illness was marked by the appearance of clinical features of a most bizarre character, and for this reason it has been thought well to record the clinical history in some detail.

PERSONAL CASE

History.—A. L., a feebleminded adult, age 21, was admitted to Leavesden Mental Hospital on August 10, 1922. A history was obtained of progressive muscular weakness commencing in his eighth year and limited, at first, to the right upper and lower limbs. For some years the loss of power was very slight and not sufficient to prevent his attendance at school. By the age of 14, weakness and wasting had made their appearance in all four limbs, and he now had difficulty in walking and was unable to undertake any form of outdoor work. In 1917 he was admitted to the National Hospital, Queen Square, under the care of Dr. Risien Russell. On his discharge from hospital he remained at home until his certification and admission to Darenth Training Colony in May, 1920. Here it was noted that he had had a fair education and that his mental defect seemed to depend on the disabilities he had suffered on account of his health, but was of a kind indicating the need for care, supervision and control.

Physical examination revealed the following: Height, 5 feet 4 inches. Weight, 6 st. Circumference of head, 22½ inches. Speech slow and deliberate. Pupils and cranial nerves normal. Weakness and marked muscular wasting in shoulder girdles, upper arms and both lower limbs. Knee-jerks absent. Plantars normal. No sphincter trouble. The patient spent all his time in a wheel-chair and was able to do useful work in the tailor's shop.

In 1924 his condition was noted to be as follows: A delicate-looking youth with only slight mental enfeeblement. He can read, write and spell simple words, but is poor at describing common objects. Mental age by the Binet Simon scale, 10 years. There is a moderate growth of hair on the upper lip and his cheeks are somewhat swollen in the position of the parotid glands; he suffers from pyorrhœa. Articulation is deliberate and slightly indistinct. Expression rather fixed and staring, with weakness of nasolabial folds.

Pupils and cranial nerves normal. No sensory disturbance. Marked wasting of pectorals and deltoids. Upper arms diffusely wasted; the head of the humerus can

be readily dislocated from the glenoid cavity by passive movement. The patient is able to write a good hand; feeds himself and occupies his time by sewing buttons on clothing. Lower limbs: marked hypotonia and muscular wasting, more on the right side. Legs too weak to support weight of body. Right heel cannot be raised off bed, nor can he turn on his side. Sitting posture can be maintained. Abdominal and plantar reflexes normal. All deep reflexes absent. Sphincters normal. His skin is dry and the areolæ of his nipples are small and oval. Blood pressure, 110/60 mm. Urine normal. Wassermann reaction negative in blood and cerebrospinal fluid. Blood: R.B.C., 4,840,000; W.B.C., 10,500; polymorphs, 65·5 per cent.; large lymphocytes, 8·5 per cent.; small lymphocytes, 25 per cent.; eosinophils, 0·5 per cent.

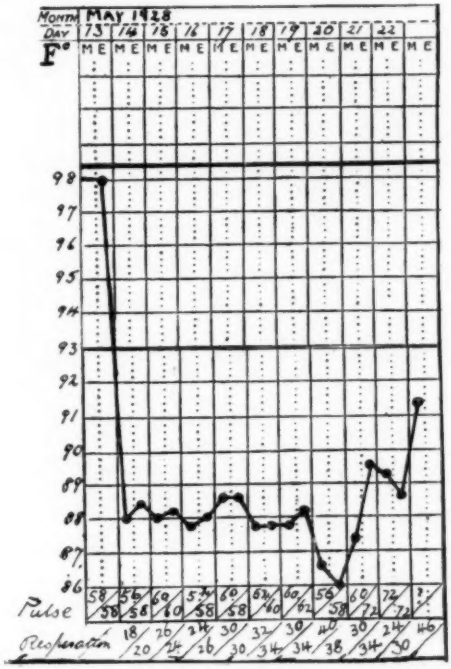


FIG. 1.—Chart of terminal phase of illness. Rectal temperature.

Progress.—There was no material alteration in his physical or mental state until December 22, 1927, when he was stated to have had a number of seizures of an epileptiform character during the night. These were accompanied by profound mental deterioration, so that he ceased to take any interest in his surroundings, became faulty in his habits and vomited frequently. From December 26 until January 7 he remained in a state of stupor with slow feeble pulse and shallow respiration, and a body temperature so low that the ordinary clinical thermometer failed to register. His general appearance resembled that of a person suffering from profound shock. On January 7 a sudden and marked improvement took place; his temperature rose to normal, his pulse became stronger and its rate, which during the period of stupor had been between

50 and 64, rose to 78. At the same time his mental state improved, and on January 18, when he was allowed up, he seemed to be quite himself again.

On May 13 there was a sudden relapse ushered in by an attack of hæmatemesis and rapid fall of temperature. On May 14 his condition was as follows: He lies in a state of stupor, groaning occasionally, not entirely unconscious, but unable to speak. Rectal temperature, 88° F. Respirations, 18, shallow and irregular. Pulse 56, small and feeble. Blood pressure, 60/40 mm. Pin-point pupils. Left oculocardiac reflex gives a rate of 44 during compression. Swallowing difficult. Profuse sialorrhœa. The skin everywhere feels icy cold and, except on the face, does not bleed when pricked. A drop of 4 per cent. cocain in the right eye caused a very slight dilatation of the pupil after the lapse of five hours. There is complete muscular relaxation, retention of urine and obstinate constipation. All superficial and deep reflexes are absent. In this state, with a rectal temperature never above 89° F. (fig. 1), he continued until May 21, when signs of bronchopneumonia were detected. The rectal temperature rose several degrees, he continued to vomit repeatedly, and death occurred at 12.30 p.m. on May 23.

REPORT OF AUTOPSY

A post-mortem examination was held three hours after death. A marked degree of muscular wasting was present in the shoulder and pectoral regions, the upper arms and the anterior aspects of the thighs. There were slight contractures at the knee joints. The fascia clothing the anterior thoracic wall was thickened and the affected muscles yellowish-white on section. Those most diseased included pectoralis major (clavicular portion less profoundly affected), pectoralis minor, serratus magnus, latissimus dorsi, deltoids, spinati, sternomastoids, omo- and sternohyoids, levator anguli scapulæ, trapezius, including upper third. Probably the triceps, biceps, supinator longus, glutei and certain muscles of the anterior aspect of the thigh were also affected, but these were not exposed. Unaffected muscles included the abdominals, diaphragm, multifidus, semispinalis, complexus, splenius capitis, muscles of the lower jaw and scalp.

Both lungs showed a condition of acute lobular pneumonia. The heart (8½ oz.) appeared normal, save for a little fatty infiltration near the apex. The liver, much reduced in size, weighed 29½ oz. and showed cloudy swelling. The kidneys were also small (3½ oz.), and the right contained a large calculus. Spleen (4½ oz.) normal. Both submaxillary glands were moderately enlarged. The pituitary, thyroid, pancreas and suprarenal glands were normal. No renal or other tumours were found.

Examination of the Nervous System.—The brain weighed 49½ oz. Slight thickening of the pia-arachnoid existed in the interpeduncular space. The cerebral hemispheres were symmetrical, large, firm and well convoluted. Scattered over the grey matter on the fore part of the brain were numerous small circular and slightly raised wart-like plaques, each a little paler than the surrounding cortex and varying in size from that of a pinhead to areas measuring 2 mm. in diameter (fig. 2). Most numerous and largest on the mesial surface of the hemisphere, they diminished in number from before

backwards, and behind the post-central gyri no plaques were found. The largest showed slight umbilication, but nowhere was there any tendency to distort the convolution pattern. The frontal convolutions did, however, show widening of the sulci, and in this situation it was obvious to the touch that there was a certain amount of diffuse sclerosis. Fewer plaques were found on the convex surface of the hemisphere, and on section no gross abnormalities were found. The walls of the lateral ventricles had a rough

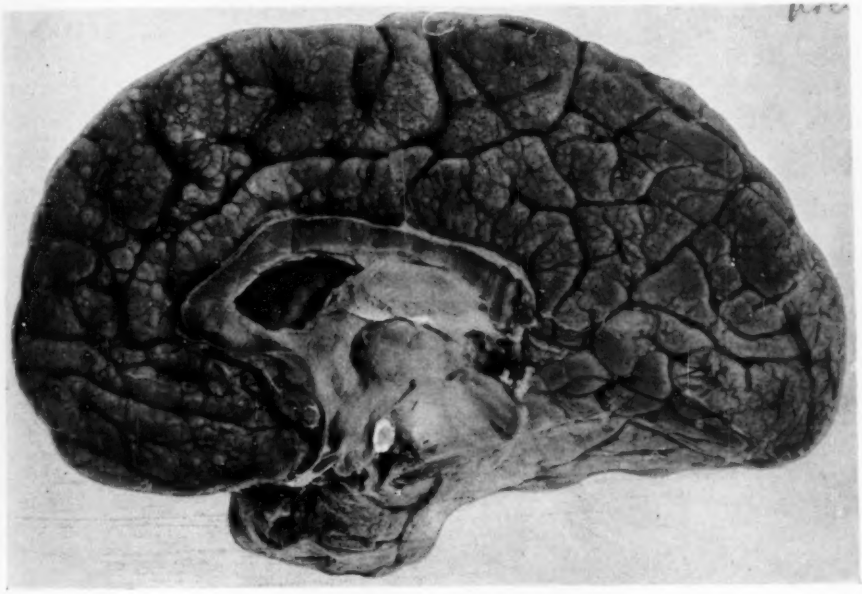


FIG. 2.—Mesial surface of left cerebral hemisphere showing the presence of wart-like plaques on the surface of the grey matter of the frontal convolutions.

uneven surface, but no tumours were present. No abnormalities were found in the cerebellum, pons, medulla and spinal cord.

Microscopic Examination.—Examination of sections taken from frontal cortex free from nodules showed a considerable amount of subpial fibrillar gliosis.

The plaques were composed almost entirely of fine neuroglial fibres, either loosely intertwined or more densely matted together with an accompanying increase of glial nuclei. Towards the deeper aspect of these masses large astrocytes were particularly numerous, and in some situations the proliferation of glial fibres seemed to be especially related to the smaller cortical bloodvessels as they entered the grey matter from the pia; in a

number of localities the appearance was as though the cortex had been drawn in by dense strands of glial tissue (fig. 3). Both in and beneath these masses—which in most instances extended no deeper than the fourth cortical layer—the normal columnar arrangement of nerve-cells was considerably disturbed, either through the actual disappearance of nerve-cells or their altered polarity; in one plaque taken from the second frontal convolution these changes were particularly well marked, the nerve-cells being twisted in

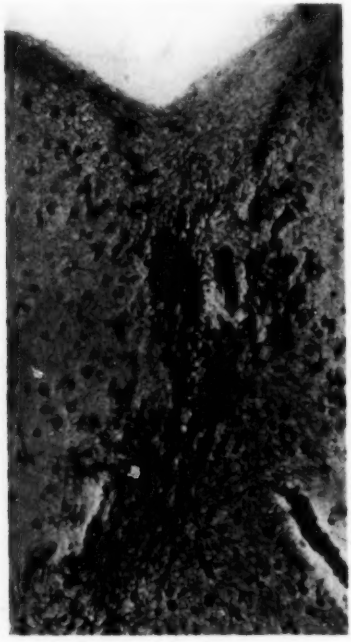


FIG. 3.—Cortical area of gliosis showing dense strands of neuroglial fibrils. Silver impregnation.

all directions (fig. 4). Here also was found a nest of elongated pyramidal cells with their long axes horizontally disposed, and at a deeper level large numbers of cells rounded in outline, possessing several short broad protoplasmic processes and large vesicular nucleus with conspicuous nucleolus; their appearance suggested developing neuroblasts (fig. 5).

Careful search failed to reveal the presence of either the large gliogenous elements or the giant multipolar cells which are found in most cases of tuberous sclerosis. The nerve-fibres entering the neighbourhood of sclerotic areas showed poorly staining and sometimes irregular myelin sheaths. The

underlying white matter showed relatively little gliosis. The bloodvessels appeared normal.

DISCUSSION

The chief interest of the clinical symptoms, particularly of those which were terminal, lies in their close resemblance to the picture of acute vagotonia. According to Eppinger and Hess,[1] in the normal individual there

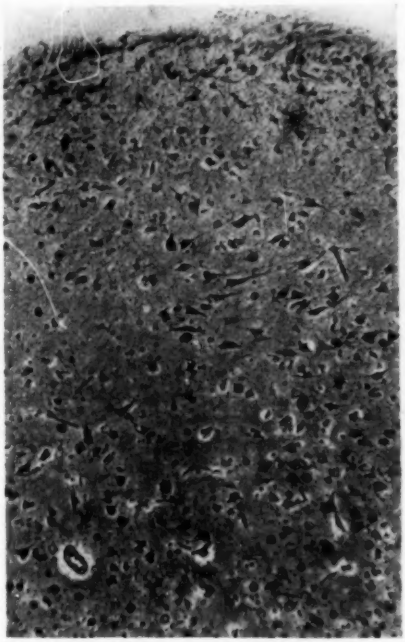


FIG. 4.—Section of grey matter of second frontal convolution showing from above downwards : (*a*) superficial gliosis, (*b*) a small group of horizontally disposed nerve-cells, (*c*) irregularly disposed neuroblasts. Silver impregnation.

exists a state of balance between the two divisions of the autonomic nervous system ; in the vagotonic subject this equilibrium is disturbed, so that a condition of increased tone is established in the parasympathetic system, which is expressed clinically by certain signs, prominent among which are acute myosis, a positive oculocardiac reflex, salivation, pallor of the skin, hypothermia and obstinate constipation.

Now these were precisely the symptoms present in the case reported above, and on the basis of the hypothesis formulated by Eppinger and Hess certain of the symptoms exhibited in mode of dying might perhaps be regarded

as a manifestation either of overwhelming hyperactivity of the parasympathetic system or of a relatively sudden failure of the sympathetic system. With regard to the existence of the former state, no evidence is to be found in the records of clinical medicine, but for the other condition there is something to be said—the symptoms of adrenal apoplexy possibly affording an example of sudden failure of the sympathetic—though, so far as the writer is aware, in no previously reported cases of muscular dystrophy have similar symptoms been observed. Nevertheless, without entering too far into the realm of pure speculation, it is perhaps permissible to suggest, as an explanation of certain of the clinical features, the possibility of a rapid failure of the

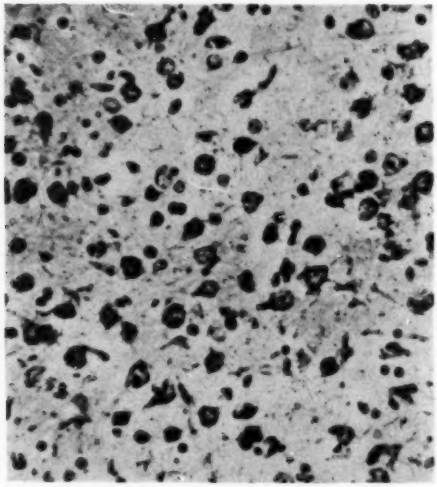


FIG. 5.—Neuroblasts from the deeper cortical layers as shown in fig. 3, but under a higher magnification. × 500. Silver impregnation.

sympathetic in this disease. On this basis, the myosis, tachycardia, vomiting and excess of salivary secretion could be regarded as due to the unopposed action of the parasympathetic, but other features of the case are not so easily explained, and the ashen pallor and coldness of the extremities might just as readily be attributed to the profound fall in blood pressure which occurred in the terminal phase of the patient's illness.

It is interesting to recall that in his Bradshaw lecture E. Bramwell[2] suggested the possibility of an implication of the sympathetically innervated elements in muscle structure as an ætiological basis for muscular dystrophy, and there can be few neurologists who have not observed in this disease clinical data indicative of neuroglandular disturbance. For the most part, spinal cord findings in previously reported cases of myopathy have been

negative, but little detailed examination appears yet to have been made of the cells and fibres of the sympathetic system. With this comparatively neglected field in mind, the study of the entire autonomic nervous system

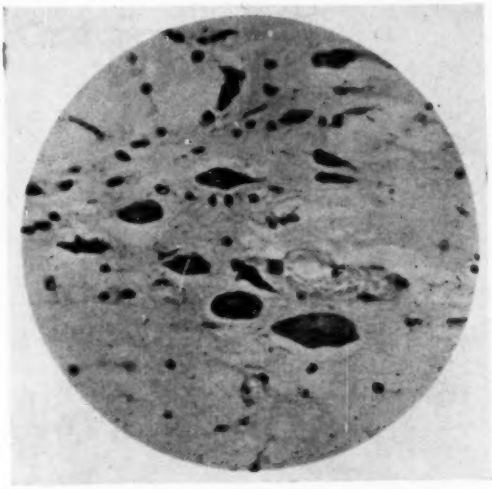


FIG. 6.—D.8 segment of cord. Intermediolateral cell column showing chronic cell degeneration. Cresyl violet stain.

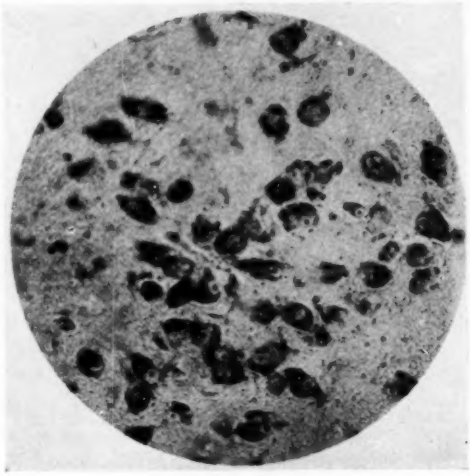


FIG. 7.—Hypothalamus. Nerve-cells from the grey matter of wall of third ventricle.

was made part of the scheme of investigation ; for various reasons the work had to be abandoned.

Reference may, however, be made to the condition of the sympathetic

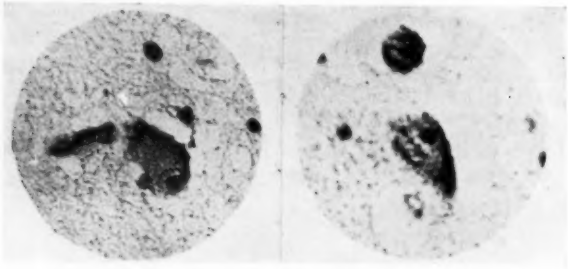


FIG. 8.—Nerve-cells in dorsal vagal nucleus. × 360.

cells of the intermediolateral cell column in segments D.8 and L.1, to those in the anterior part of the hypothalamus and to the cells of the dorsal nucleus of the vagus. In each of these situations evidence of chronic cell shrinkage with lateral displacement of the nucleus was obvious (figs. 6, 7, 8), but a

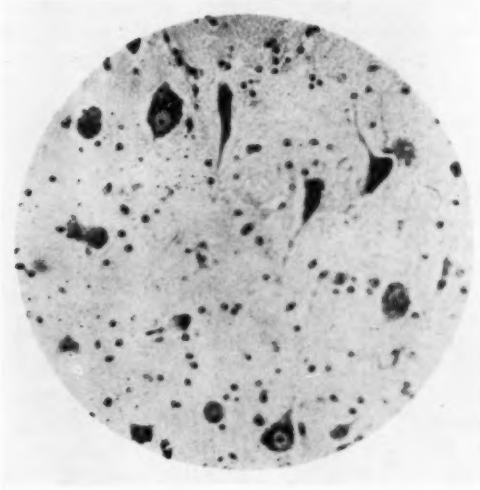


FIG. 9.—Spinal cord. D.1. Anterior horn cells.

comparison of these cells with the larger anterior horn cells of the cord (fig. 9) showed that the neuronic degeneration was widespread and not limited to those concerned with the activity of the autonomic nervous system.

Lastly, it is necessary to mention the enlargement of the salivary glands which was present in this case. To avoid disfigurement of the body, sections were taken from the submaxillary and not from the parotid glands. No obvious changes were noted, and the significance of this enlargement remains obscure. Sialorrhœa was a striking feature in the clinical course, but it was more or less terminal and the salivary enlargement preceded it by some years without excessive secretion, so that the enlargement could hardly be attributable to a long-continued state of autonomic stimulation. The condition appears to have been only once previously observed. Under the title 'A case of Erb's juvenile dystrophy associated with bilateral enlargement of the

FIG. 10.—Appearance of cortical plaque. Note its limitation to the superficial layers of the grey matter. Silver impregnation.

parotid and submaxillary glands,' Mitchell Clarke [3] in 1903 reported the presence of symmetrical enlargement of the salivary glands in a youth of 18. The onset of the muscular atrophy and swelling was simultaneous, a coincidence which suggested to the author the possibility of some common cause for the two conditions.

CORTICAL CHANGES—CEREBRAL HEMISPHERES

The most striking naked-eye change consisted in the presence of several hundred small, circular, raised wart-like plaques which seemed to be on, rather than in, the substance of the grey matter (fig. 10). Particularly numerous on the frontal convolutions, they were much fewer on the more caudally placed convolutions, and none was found on the occipital convolu-

tions. The majority of plaques were about 1 mm. in diameter and the largest were little more than twice this size. All were readily recognized by inspection even after prolonged formalin fixation. In addition, on the mesial surface of the frontal lobe indication of a diffuse gliosis was shown by narrow gyri separated from each other by deep fissures. On section, the cortex showed no loss of the sharp line of demarcation which normally separates grey matter from white, the sclerotic process involving principally the upper layers and leaving the architecture of the deeper portion of the cortex undisturbed. The walls of the lateral ventricle showed slight irregularity but no ' candle guttering ' or infiltrating tumours of the type seen in tuberous sclerosis.

These appearances are in marked contrast with those encountered in tuberous sclerosis. In the latter condition, the hypertrophic convolutions and sclerotic nodules are more easily felt than seen, and in hardened brains are extremely difficult to pick out; in diameter they are seldom less than $\frac{1}{2}$ cm., and are not infrequently very much larger. Their distribution is without reference to any particular cortical region, and when sectioned it is seen that they blend with the underlying white matter.

In its microscopical characters the case reported here showed two points of similarity—a reaction of the surface neuroglia and disturbance of cortical architecture; here the resemblance ended, and numerous points of difference were obvious. The raised wart-like nodules occupying only a portion of a convolution presented an appearance quite unlike that seen in tuberous sclerosis; the transition between normal and pathological fields was not gradual but abrupt, and usually took place in the superficial cortical layers. Groups of atypical nerve-cells resembling neuroblasts were encountered in the superficial strata of the cortex, but a diligent search failed to reveal the presence of any of the remarkable large elements which characterize tuberous sclerosis.

On the whole, the disturbance of cortical architecture was more widespread but much less severe than in the latter condition, and the macroglial reaction decreased in a gradual and progressive way as one followed the convolutions from the frontal to the occipital poles.

Gliosis as a developmental abnormality is by no means an uncommon finding in the brain of the mental defective, and a study of the microscopical appearances in this case suggests that we are here dealing with an unusual type of generalized glial hyperplasia limited to the grey matter of the cerebral cortex and probably congenital in origin.

SUMMARY

A feebleminded male with Erb's juvenile type of muscular dystrophy died after the disease had been in existence for 20 years. During life the patient exhibited a bilateral and symmetrical enlargement of the salivary glands. The terminal illness was ushered in by a marked and persistent hypothermia, bradycardia, low blood-pressure, profuse salivation, repeated

vomiting, myosis and a positive oculocardiac reflex; it is suggested that a sudden failure of the autonomic nervous system may have caused these unusual terminal symptoms.

Pathological examination confirmed the diagnosis of muscular dystrophy, and, in addition, revealed the presence in both cerebral hemispheres of a special type of glial hyperplasia; this took the form of small, raised wart-like nodules in the cortical grey matter, numerous in the frontal convolutions and diminishing in number towards the occipital poles, in many situations accompanied by a disturbance of cytoarchitecture and by the presence of primitive nerve-cells. No glial or neuronic cells of giant type were found and the visceral organs contained no tumours.

The condition appeared to represent an unusual type of primary gliosis rather than an abortive form of tuberous sclerosis.

REFERENCES

[1] Eppinger, H., and Hess, L., *Vagotonia*, 1915.
[2] Bramwell, E., *Lancet*, 1925, **2**, 1103.
[3] Mitchell Clarke, J., *Brain*, 1903, **26**, 202.

Editorial

CLINICAL RESEARCH IN PSYCHIATRY

IT is not so long ago that there was a lively discussion in this country on the scope for clinical research in general medicine. There can be few branches of clinical medicine in which there is more scope remaining for pure clinical research than in psychiatry. It is a curious reflection that few discoveries of fundamental importance in psychiatry have come from the laboratory, apart from the work that led to the delimitation of the structural diseases of the brain—especially of general paralysis. But neither in manic-depressive psychoses nor in the large miscellaneous groups that we call the schizophrenic, nor in the paranoiac conditions, and still less in the psychoneuroses, can anyone point to anything fundamental in our understanding of these conditions that has been due to the technique of the laboratory, and that is at the same time widely accepted. Even in epilepsy clinical observation has yielded so far the most important results. Only in mental defect and the miscellaneous group of mental illnesses which are secondary to gross bodily disease are we indebted to other than clinical methods for our deepest understanding of the conditions involved.

This is a state of affairs which can hardly fail to excite comment, but judging from the relative proportion of papers devoted to clinical and laboratory work on psychiatry in this country one would imagine that the circumstance had passed unobserved. This is not to suggest that laboratory work should be in any way relaxed. It may be that with the better methods now increasingly at the disposal of the biochemists the disciples of what is after all comparatively a young science may at no distant date find it possible to come to some discoveries worthy to be regarded as fundamental. But in the meantime the lessons of the past point in the direction of the benefit to be obtained from a more intensive pursuit of purely clinical methods, pari passu with laboratory studies. This is the more to be welcomed as, after all, the training of most of the men who take up psychiatry is predominantly clinical. Doctors in general are not specially trained for research on

physicochemical lines. In so far as their training has been mainly clinical they have, in 99 cases out of 100, received a training which qualifies them better to carry out clinical investigations than to do any other kind.

A young graduate taking up research, if he wishes to work in a laboratory, must in these days master a technique and must usually for a considerable time confine himself, if he hopes to make a contribution, to a narrow field in which that technique can be used once he has acquired sufficient skill in it. It is exceptional for a man to have had the additional qualification of a long training in some branch of pure science. It would consequently appear that his best work is likely to be done in that domain for which his training fits him more than any other, namely, clinical work.

It will be asked in what directions clinical investigation remains to be done. One obvious line of inquiry is the psychopathology of the psychoses, which would repay systematic analysis not only for the sake of the psychoses themselves, but for the sake of confirming or modifying current psychopathological theories. In the psychoses it is commonly held that much that is usually unconscious comes to frank conscious expression. What would take weeks or months of effort to obtain, if it were obtainable at all by free association in other circumstances, is here to be observed in return for the trouble of patient selection of the data which present themselves to direct observation. This should be of value for the understanding of the type of psychosis in question, independent of any causal claim of psychological factors in them. Even if it cannot be shown that such factors are pathogenic, the value of elucidating them for the dynamogenetic understanding of the personality and in demonstrating along what lines the personality can disintegrate should be very considerable. In other words, the principle which Hughlings Jackson applied to the study of delirium might be fruitfully applied to the study of the psychoses, especially now that there exists, as there did not in his time, a body of psychopathological theory with which the results of clinical observation may be compared.

It will be admitted that there is still much scope for the introduction of accurate methods into clinical psychiatry. The possibility of supplying statistical analysis to the delimitation of various syndromes suggests itself. For example, in what we call manic-depressive psychoses on the one hand and schizophrenic psychoses on the other, what is the intercorrelation of the various symptoms? What percentage of depressions are

retarded ? How does the retardation correlate with hallucinatory mental experiences or with delusions of sin, or more generally and less superficially with indications of feelings of guilt ? Or what features of depressions in general correlate specially highly with retardation ? This is a sample of the kind of question that might be answered by systematic clinical enquiry along statistical lines.

The most obvious field for statistical investigation however is that of prognosis. There are few collections of cases large enough to give statistical validity to statements about outcome and still less ample are the data for correlating particular symptoms with prognosis. For example, there is little statistical basis for the clinical impression psychiatrists often have of the gravity or otherwise of any particular symptom. Do unreality feelings in depression really betoken a prolonged attack ? Is a single symptom like the fear of insanity expressed in the course of an agitated depression ominous of a prolonged course ? Then as for recurrences, what is the proportion of manic-depressive patients having a third attack ? There has been a study of this last topic by Pollock in the New York State Hospitals, but we know of no similar study in this country.

Closely related to the study of prognosis is the problem of the advisability of what are usually called ' follow-up ' studies. These have not often been done hitherto and it is sometimes objected that they are necessarily unreliable, and even that they are best left undone. This last contention is too extreme to be considered, but it is based on the undoubted fact that there are many fallacies in a ' follow-up.' No fallacy however is greater than that of supposing that because a patient leaves hospital symptom-free he is necessarily cured. Nowhere else is this more likely to be misleading than in mental diseases, which are often by definition the reaction of an individual to a continuing environment to which he must return. A cure ' in vitro ' is not, so to speak, necessarily a cure ' in vivo.' It is held that the following up of a patient who has suffered from some form or other of mental illness does not give a true account of his condition. His criteria of recovery may not be those of his doctor or his wife. While a follow-up by correspondence may spell difficulties of this kind, they are largely overcome by a personal enquiry carried out by a trained person. It is not to be expected that even by these means the intimate data can be obtained which will give a complete picture of the patient's mind. Science and the interests of the patient are not always

at one in this matter. But it will always ensure that social recovery is assessed with reasonable accuracy.

Controls are much needed, especially when claims are made for forms of treatment. For example, what proportion of psychoneuroses recover spontaneously? How do those treated compare with those untreated in the degree and nature of their recovery?

It will be seen that what is advocated is not a descriptive study of symptoms, which has been done often enough, but clinical observation with a view always to finding out what the symptoms mean.

Abstracts
Neurology

NEUROPATHOLOGY

[43] Striatal localization in chronic progressive chorea.—M. NEUSTAEDTER. *Jour. Nerv. Ment. Dis.*, 1933, **78**, 470.

A REVIEW of the literature of this subject is given in respect of both focal choreas and chronic progressive choreas. Three cases with necropsies are described—two cases of Huntington's chorea and one of arteriosclerosis.

Macroscopically, all cases showed convolutional atrophy involving the frontal and central gyri and a corresponding diminution of the white substance. In two cases the cortex and white substance of the rest of the brain were considerably shrunken, while in the third these parts were fairly well conserved in volume and consistency. There was a moderate dilatation of the lateral ventricles, particularly of the anterior and posterior horns in the first two cases, but no true hydrocephalus.

The caudate nucleus and putamen were atrophic in two; in the third these nuclei were fairly normal in size. The globus pallidus was considerably shrunken in the first two cases only.

Microscopically, the cortex in the first two cases showed unmistakably that the cytoarchitectonic arrangement of the third and fourth layers was in obvious disorder. In the third case all layers presented this disarrangement and in addition many areas of softening. If these cortical changes in the frontoparietal areas are responsible for the mental defect, and the author is willing to subscribe to this theory, the mental deterioration in all three cases certainly goes hand in hand with their pathology.

Changes in the small cells of the neostriatum of the first two cases were of a chronic degenerative type, while those in the third were of an acute type due to arteriosclerotic changes with a great many areas of softening. The large cells in all cases were fairly well preserved. The pallidal cells were not involved in any case. In the third, the thalami, red nuclei and olives were markedly affected. The cell destruction was of a chronic nature antedating that of the neostriatum. The changes of the cerebellum in the first two cases were relatively slight and then only in a few places. In the third the dentate nuclei showed considerable cell destruction of a chronic type.

If the changes in the thalamus, red nucleus or cerebellum were solely responsible for the choreiform syndrome it would certainly have been evident in the third case long before its noted appearance. As an inmate of a home, the patient was observed for several years before her admission to the hospital, and according to trustworthy reports she was to all intents and purposes in a

fair state of health. Six months before admission choreiform movements of the head and much later of the facial muscles were observed, and two months later the extremities became involved. From this time on she became progressively and rapidly worse.

In view of the pathology of the third case, and the lack of involvement of the thalami, red nuclei, olives and cerebellum in the others, the author claims that the assumption is justified that in all three cases the choreiform syndrome was probably due to the destruction of the small cells of the neostriatum.

R. G. G.

[44] **Contribution to the study of whoopingcough encephalitis** (Contributo allo studio delle encefalopatie da pertosse).—L. MENCARELLI. *Riv. di pat. nerv. e ment.*, 1933, **42**, 552.

THE brains of three children who in the course of attacks of whoopingcough developed various complex symptoms referable to the central nervous system were examined. The various theories are referred to and especially the question of the pathogenesis of the hæmorrhagic lesions that were found. The literature of this subject is thoroughly reviewed and a comprehensive bibliography is given.

R. G. G.

[45] **Studies of pathological neuroglia in man** (Studio sulla nevroglia patologica nell' uomo).—G. CANZIANI. *Riv. di pat. nerv. e ment.*, 1933, **41**, 633.

USING the silver stain of Lugaro the author has examined the protoplasmic neuroglia. He compares two cases of non-treated general paralysis, one acute and one chronic, with two treated by malaria, and two with the sulphur pyretic treatment. In all cases the neuroglia exhibited a pathological appearance, and the lesions showed themselves to be definitely progressive. In the malarial cases the neuroglia was slightly less pathological, since although definitely hypertrophied it showed less tendency to proliferation and fibrous transformation. In the cases treated by sulphur the neuroglia manifested no lessening of pathological characteristics as contrasted with the usual course of the disease.

R. G. G.

[46] **A study of the cerebral glia at a distance from cerebral tumours** (Studio sulla glia cerebrale a distanza dai tumori encefalici).—F. CARDONA. *Riv. di pat. nerv. e ment.*, 1933, **41**, 279.

A DIFFUSE glial reaction all over the cerebrum has been found in cases of cerebral tumour, in the form of hypertrophy and hyperplasia at a distance from the tumour. The author considers that the most important factor

accounting for this reaction is the toxicity of the tumour, and points out the importance of the appearance of this glial reaction in cases of cerebral tumour in post-mortem examinations.

R. G. G.

[47] **Histopathological findings in two cases clinically diagnosed dementia præcox.**—Armando Ferraro. *Amer. Jour. Psychiat.*, 1934, **13**, 883.

In the first case an encephalopathy was found bearing the ear-marks of a toxic process and consisting of patchy demyelination with replacement of the disintegrated tissue by proliferated glia fibres and concomitant involvement of the cellular elements of the cortex, particularly in the frontal, central and temporal areas. In the second case the process was one of diffuse sclerosis, and the pathological changes were severe though unaccompanied clinically by neurological manifestations. The reason for presenting these two cases is to call the particular attention of clinicians to the fact that cases of so-called functional psychoses exist in which the clinical manifestations of any organic component may be lacking, and that such cases may nevertheless have as a basis a definite pathological process of the brain. Attention is also drawn to the fact that in clinical diagnosis reliance should not be placed too much on the existence of the so-called organic type of reaction, as this may be absent clinically though a definite organic pathological process is present in the brain. Hence it is not safe to assume that at the basis of dementia præcox no organic disease will be found.

C. S. R.

[48] **The behaviour of urea, uric acid and cholesterol in the blood in diseases of the meninges and brain** (Il comportamento del tasso ureico, dell' acido urico e del colesterolo del sangue nel corso di meningo-ed encefalopatie).—N. Lolli. *Riv. di pat. nerv. e ment.*, 1933, **41**, 96.

The author has examined the biochemical changes in the urea, uric acid, and the cholesterol in the blood in the course of meningeal and encephalic diseases, and points out that in many cases it is altered with a tendency to rise above the normal, sometimes markedly. The modifications in the rate of the urea have nothing to do with that of the chlorides, with elevation of temperature or renal alterations.

To check these results he has experimentally punctured the floor of the fourth ventricle in rabbits and has obtained the same modifications ; in some cases these modifications have been obtained also by means of lesions of cortical regions, but probably they are to be explained as being caused by the excitement at a distance of the regulative centres of metabolism, which the author, in accordance with his investigations, is inclined to localize in the floor of the fourth ventricle.

R. G. G.

SENSORIMOTOR NEUROLOGY

[49] **The effect of intercurrent chronic pulmonary tuberculosis on the convulsion threshold in epilepsy.**—I. J. KARLSBERG. *Amer. Jour. Psychiat.*, 1934, **13**, 799.

INVESTIGATION seems to show that epilepsy does not predispose to chronic pulmonary tuberculosis, or vice versâ. In a series of 100 cases of epilepsy with such intercurrent tubercle in its active stages the frequency of seizures was markedly decreased in the majority of cases, with a demonstrable loss of weight. A review of the literature reveals that there is a reduced metabolic rate in epilepsy and a definitely increased rate in pulmonary tubercle, that convulsions are often absent in diseases with a high blood sugar such as diabetes and cancer, and that hyperglycæmia tends to occur in the tuberculous individual; that a normal blood-serum calcium is usual in epilepsy and that it is often found to be elevated in pulmonary tubercle, that a reduction in the sodium chloride of the blood of epileptics often reduces the frequency of convulsions, and that a constantly low blood chloride is often observed in patients with active pulmonary tubercle; that comparisons of the blood pictures in epilepsy and in pulmonary tubercle show the occurrence of hypocholestæmia and an increased rate of sedimentation of the blood in both conditions; that the *p*H values of the blood seem to indicate a tendency in the epileptic to approach a more alkaline reaction, and in the tuberculous patient a more acid reaction. From the above data suggestions are made as to the mechanism involved in active chronic pulmonary tuberculosis in raising the convulsion threshold in pre-existing epilepsy so that the frequency of seizures is noticeably reduced.

C. S. R.

[50] **The gluteus maximus reflex is a true reflex** (Il riflesso del muscolo gluteo grande e un vero riflesso).—G. DALMA. *Riv. di pat. nerv. e ment.*, 1932, **40**, 567.

THIS thesis is held to be true in virtue of the demonstration that in a case of traumatic spinal compression involving the posterior roots of the 4–5 lumbar segments, the reflex was abolished, while in a case of spondylitis in the dorsal region causing injury to the pyramidal tracts, the reflex was increased so that the gluteus exhibited clonus. The reflex is therefore a true one involving a spinal arc and is not an idiomuscular twitch.

R. G. G.

[51] **Idiopathic multiple spontaneous fracture (Lobstein's disease) in epilepsy** (Osteopsatirosi idiopatica o malattia di Lobstein in epilettico).—J. NARDI. *Riv. di pat. nerv. e ment.*, 1933, **41**, 1.

THE author describes a case in detail and then argues that the pathogenesis of the osseous dystrophy might be the same as that of the epilepsy on the

basis of a deficiency in calcium, which was present, and therefore possibly responsible for both. The deficiency he attributes to an endocrine anomaly due to lesions in the region of the diencephalon and hypophysis.

R. G. G.

PROGNOSIS AND TREATMENT

[52] **Tryparsamide in the treatment of neurosyphilis.**—H. H. Reese. *Jour. Nerv. Ment. Dis.*, 1933, **78**, 354.

A discussion of the usefulness of tryparsamide in various types of neurosyphilis and a report of results. In a group of 341 cases of general paralysis clinical arrests or remissions were obtained in 54 per cent. In 306 cases of meningovascular syphilis clinical or serological cures were obtained in 78 per cent. In paresis a combination of tryparsamide and malarial therapy will be found to be desirable.

R. G. G.

Psychopathology

PSYCHOLOGY

[53] **Some normal odour effects and associations of psychoanalytic significance.**—Donald A. Laird. *Psychoanalytic Review*, 1934, **21**, 194.

Data from 254 distinguished living persons show that the sense of smell still occupies a prominent place in normal mental dynamics and has not fallen into the state of disuse commonly imagined. Of most significance in these data are the emotional ' drives ' released by certain odours and the symbolism in many of the odour-linked memories and experiences of these accomplished and apparently non-psychotic persons. Regression to childhood odours, giving rise to nostalgia and a poignant longing for the past, was reported. Other brief reports given show varied trends. Some questions are raised by the fact that of many experiences with the romantic element in odours only a small majority are from women. This in spite of the data showing that memories stimulated by odours are more frequent and vivid in women than men. As far as these spontaneous accounts show, artificial, or perfume odours seem to be more potent in sensual stimulation than natural body odours. The relative rarity of olfactory abnormalities should be looked upon as reflecting the evolutionary dictum that ' the oldest neurologically is the most stable.' The common occurrence of psychoanalytic mechanisms here reported shows the active rôle of the sense of smell in human mental life.

C. S. R.

[54] **Psychological weapons in animal fight.**—R. W. G. Hingston. *Character and Personality*, 1933, **2**, 3.

Animals fight psychologically as well as physically. The fact has scarcely been noticed by naturalists. When we come to understand it properly, it provides us with a simple explanation of the meaning of their colours, adornments, courtships, moults, songs, extravagant weapons, differences between their sexes, and other fundamental problems. It in fact explains in terms of battle everything that Darwin included under his hypothesis of sexual selection.

An enraged animal when on the point of attacking another tries to make itself look as terrible as possible. A dog raises his hair and exposes the canine teeth, the gorilla lifts the crest on his scalp, the lion spreads his mane, the mongoose spreads the hairs on his tail, etc. These weapons exist everywhere in nature. They assume a multitude of forms and operate in the most varied ways. Instances are given in birds, fishes, serpents, frogs, and insects. Animal colour, too, has this function. Bright colour is part of an animal's fighting machinery and renders it more effective. Conspicuous hairy growths are also in essence fighting machinery and this is applicable to man. In all sections of animal creation colour fulfils two conflicting functions. One half of the pattern has a concealing function, the other half has an advertising function in that it serves to threaten the adversary. The actual pattern that an animal possesses is the result of a compromise between these functions. The two patterns are the outward expression of the dominant emotions of fear and anger which are in opposition. It is almost invariably the males that have the brighter tints because this sex requires more threatening machinery for fighting. As winter is a peaceful season birds moult and throw off their bright colours and resume them again in the summer when fighting is often needed for defence of territory and nest. Since sex is regarded as essentially a hostile act, the colour pattern is of import in courtship behaviour in the same way as woman is attracted by signs of virility and strength.

C. S. R.

[55] **The Rorschach ink-blot test.**—Philip E. Vernon. *Brit. Jour. Med. Psychol.*, 1933, **13**, 271.

This test constitutes a novel approach to the diagnosis of personality which has been but little explored outside Switzerland and Germany. The test consists of ten standard blots, some black and grey, some coloured, which are examined in turn by the subject under natural conditions. He states what each blot or part of a blot suggests to him, without time limit or other restrictions, and the experimenter records his responses or interpretations. The test can only be applied individually. The chief use of it is said to be in the clinical diagnosis of mental diseases. In schizophrenia, cycloid or organic

psychoses, epilepsy, etc., characteristic scores should be obtained on the various categories of response. Few of these have received any quantitative validation. Many psychiatrists have attested the value of the method for the insight which it gives into the total personalities of their subjects, normal or abnormal. The writer states that he does not know any other test of personality or temperamental traits which will tell him as much about his subjects in so short a time as does the Rorschach test.

C. S. R.

[56] **A note on humour.**—Lucile Dooley. *Psychoanalytic Review*, 1934, **21**, 49.

Freud affirms that when an individual treats a situation humorously he treats the person involved, whether it be himself or another, as a child. There is a displacement of cathexis from ego to superego—the person lives more as a superego than as ego—when he is humorous. To this greatly expanded superego the ego appears as infinitesimally small, its dangers and sufferings consequently laughably insignificant. Humour, therefore, is one of those methods which the human mind builds up in order to escape suffering. Like neuroses and intoxications, humour renounces reality and permits phantasy to take its place, but unlike these other methods of defence the escape is secured at no cost to mental health, with no break in mental integrity, because in humour there is no withdrawal of the actual painful content from consciousness. When the energy of threatening instinctual drives of a sadistic or masochistic tendency is displaced on to the superego, the latter can thus allow pleasurable activity to the ego, since this activity is now desexualized and free of aggression. From this triumph of narcissism, which is the essential characteristic of humour, and from this spiritualization, humour derives its lofty, uplifting and benevolent character. The humorous attitude has two invariable characteristics which serve to preserve narcissism: first, its emphasis on and pleasure in littleness, the 'little thing' being really the ego, which is not only spared suffering by this reduction, but is given narcissistic pleasure by the glimpse of the outer world in pygmy proportions; and, second, its indulgence of phantasy—also childish—which clothes the hardness of reality with comforting playfulness. Unlike wit, humour is never sadistic. It is an attitude towards life that tends constantly to turn aside suffering. While it may not give the intense pleasure or produce the instant laughter evoked by wit, it serves to create happiness, which is less rich but more continuous than pleasure. We should expect to find the first indications of humour in that phase of psychic development where the superego has its beginnings. Full-fledged humour is probably post-adolescent but no character trait comes forth suddenly full grown.

C. S. R.

PSYCHOSES

[57] **The acute schizoaffective psychoses.**—J. KASANIN. *Amer. Jour. Psychiat.*, 1933, **13**, 97.

A GROUP of nine cases is presented in which there is a blending of schizophrenic and affective symptoms. The psychosis is characterized by a sudden onset in a setting of marked emotional turmoil with a distortion of the outside world and presence of false sensory impressions in some cases. It lasts a few weeks to a few months and is followed by a recovery. The patients were young people, in the twenties or thirties, in excellent physical health, in whom there was usually a history of a previous attack in late adolescence. The pre-psychotic personalities of the patients showed the usual variation found in any group of people. A good social and industrial adjustment, the presence of a definite and specific environmental stress, interest in life and its opportunities, and the absence of any passivity or withdrawal are some of the factors favouring recovery.

C. S. R.

[58] **Organic functions in schizophrenia.**—R. G. HOSKINS and F. H. SLEEPER. *Arch. of Neurol. and Psychiat.*, 1933, **30**, 123.

AN epitomized account of the results of a study of the physiological functions in schizophrenia as brought out by repeated tests on 57 male subjects over a period of seven months. An illustrated case is included. Grossly, in most respects the patients were organically normal. They averaged 16 per cent. under-weight and as a group showed a high incidence of poor circulation of the skin, irregularities of the pupil, abnormal reflexes, and bad teeth. The blood pressure, rate of oxygen consumption and, to a slight extent, the pulse rate were basically depressed. Hypometabolism was a characteristic condition. The urinary constituents were normal in amount, as was the distribution of the nitrogenous components, but the total volume was twice the normal amount and the variability of output three times as great as in a control series. This suggests abnormal functioning of the diencephalon or of the posterior lobe of the pituitary gland. The galactose tolerance was notably variable but the blood sugar during fasting was strictly normal in both average and range. The blood pictures were characterized by a high incidence of moderate secondary anæmia and leucocytosis. The chemical constituents of the blood were unusually variable in individual cases but the averages were mostly normal. The cholesterol content ranged slightly low. It is concluded that as regards homeostasis, schizophrenia is characterized by two deviations from normality. The ability to maintain the ' steady state ' is diminished and some functions are basically displaced in an upward, others in a downward, direction.

C. S. R.

[59] **Psychoses associated with childbirth.**—D. N. Parfitt. *Jour. of Ment. Sci.*, 1934, **80**, 43.

This paper is based on the case-histories of 14 successive patients admitted to a mental hospital. Of these, 11 were puerperal, and three were psychoses arising during the course of pregnancy. Eleven patients recovered sufficiently to be discharged, two died, and one remained in hospital. The various factors which apparently influenced the course of these psychoses are investigated. In all but one patient albumen was found in the urine soon after admission. In six the amount was considerable and in three it persisted during the hospital stay. Two of these latter had casts as well. In the remaining seven the albumen disappeared in a short time. Pus cells were persistent in three cases, and *B. coli* was cultured from the urine of eight. The urine of the other six was sterile. Four patients had indican, two acetone and two sugar. Water excretion tests as tests of renal function proved of little value, but the excretion regularly increased with improvement in the mental health, and it was found possible to correlate the increased excretion figures with reduction in the emotional tension. It is noted that the estimation of blood cholesterol has little clinical use in the treatment of psychoses associated with childbirth. Urea excretion tests also proved of no value.

Septic infection was fairly frequent and two patients died of septicæmia. Subinvolution was present in eight of the remaining 12, and in six of these the lochia were offensive, while four patients had temperatures above 100° F. In a few focal sepsis was in evidence in the teeth, tonsils, and sphenoidal sinuses. Anæmia was a common finding and seven had hæmoglobin percentages below 60, the average of these being 48 per cent. Abnormal neurological signs were practically confined to changes in the pupil reaction and the tendon reflexes. The pupils were dilated in six cases, normal in five, sluggish in seven, and contracted in none. The reflexes were normal in four, increased in six, and exaggerated in four. One patient certainly, and another possibly, had deficient thyroid secretion. It is pointed out that the possible rôle of posterior pituitary hormone as a cause of puerperal infection should be borne in mind when stimulating the uterus in puerperal cases.

The diagnosis in this small group was: schizophrenic states, six (hebephrenic three, paranoid two, simplex one); manic state, five; depressive state, one; toxic confusional state, one; stupor, one. The sexual life, both pre-psychotic and later, is discussed.

C. S. R.

[60] **Plasma lipoids in mental depression.**—David Slight and C. N. H. Long. *Amer. Jour. Psychiat.*, 1933, **13**, 141.

This study indicates that a comparison of psychotic depressions of the manic-depressive group with normals shows in the former: (1) The total fatty acids of the plasma are higher in the fasting state; also that after a fat meal

there is a greater rise in the total fatty acids than in normal subjects. (2) The cholesterol is at a higher level, both in the fasting state and after a fat meal. (3) The iodine number is lower in the depressives, which can be taken to indicate that in these cases there is a greater proportion of saturated plasma lipoids.

C. S. R.

PSYCHOPATHOLOGY

[61] **Mental symptoms in lesions of the frontal lobes** (Ueber psychische Störungen bei Stirnheinerkrankungen).—A. KNEPEL. *Allg. Zeits. f. Psychiat.*, 1933, **100**, 377.

THE author reviews the different symptoms that have been emphazised as of importance at one time or another in the diagnosis of frontal lobe lesions. On the purely neurological side he discusses the so-called 'frontal ataxia' of Bruns—difficulty in turning the head and trunk to the opposite side; the tendency to walk towards the side of the lesion (Barány); nystagmus to the opposite side and abduction of the limbs towards the same side (Rothman and Blohmke); changes in muscular tone and a peculiar ipsilateral tremor of the extremities (Beevor and Stewart). He further mentions the return of some primitive reflex mechanisms—Pötzl's paradoxical flexion reaction and the defence reaction of Magnus—the two combined recalling the gait of quadrupeds. Among other neurological signs mentioned are a 'pseudo-Kernig's sign,' conjugate deviation of the eyes, epileptic attacks of Jacksonian type, sphincter disturbances, aphasia and agraphia. Akinetic phenomena closely related to the mechanism of will power are emphasized.

As regards mental symptoms the following are described; amnesia in speech, monotonous and monosyllabic speech, spontaneous mutism, 'telegram style' of speech. Akinesia of body movement together with apathy and disturbances of thought and orientation sometimes suggest catatonia and may lead to an erroneous diagnosis of schizophrenia with stupor. Lack of interest in surroundings, disorders of attention and of memory and orientation lead to important changes of personality, which together with mental sluggishness, loss of energy and self-control often usher in the disease.

The author's personal cases had in common a progressive onset with mental changes of an indefinite character. The lack of spontaneity, apathy and akinesia were marked features. In speech, this lack of spontaneity found expression in spontaneous mutism, or a monosyllabic and monotonous voice without any signs of aphasia. The akinesia in the motor sphere gave rise to very marked reduction of spontaneous movements although there was no actual paresis at that stage of the disease. No localizing value is attributed to this latter phenomenon.

In connexion with the mental apathy, the tardiness of all intellectual

processes is commented upon. In only one case was an inclination to jocularity and slight euphoria observed.

The author's observations indicate that absence or presence of papillœdema has no real diagnostic value and that gliomas of the frontal lobe give rise to papillœdema in four-fifths of the cases, excepting those of very slow growth and those situated at the base of the brain. Meningiomas situated on the floor of the cranium anteriorly do not give rise to papillœdema whereas those of the cerebellum or the falx cerebri are usually accompanied by it.

In an analysis of the different symptoms, stress is laid on the important fact that tumours with identical localization may produce different symptoms. It is argued that the clinical picture depends on several factors amongst which prominence is given to: (1) variety of tumour (glioma, meningioma, etc.); (2) rate of growth—slow, rapid; (3) constitution and age of patient (mental changes more prominent in old people); (4) the side affected (preponderance of akinesia in left-sided lesions).

Integrity of the left frontal lobe, according to this author as well as to many other observers, is of primary importance for the uninterrupted normal functions of motor and psychic activity. In connexion with this fact an attempt is made to throw light on the much disputed question of the specific functions of the frontal lobes. The author recalls the so-called frontal lobe hypothesis of Meynert and Flechsig (the frontal lobe region as the seat of higher intellectual processes, the organ of thought and of the association of ideas). This theory has now been discarded by most observers although some pathological and embryological findings appear to support it. The theory that the higher mental processes are localized in any particular group of gyri in the brain seems no longer tenable as more recent observations and experiments tend to make the whole cortex responsible for higher intellectual activity.

The author concludes his paper by tentatively putting forward the hypothesis that as the integrity of the frontal lobes is a sine qua non for the normal functioning of the higher intellectual processes, there must be some centres which regulate impulses reaching the frontal lobes from other parts of the brain.

L. Z.

[62] **Considerations for evaluating the galvanic skin reflex.**—Chester W. Darrow. *Amer. Jour. Psychiat.*, 1933, **13**, 285.

Work in the past has failed to have the value which had been expected. The first and most general principle which may be laid down is that a high galvanic reactivity seems to be characteristic of good physical health. This is probably attributable to the fact that subnormal physical states and febrile conditions are generally attended by a subnormal secretion of sweat.

On the other hand, an extreme hyperidrosis which presents pathology in the opposite extreme is likewise generally accompanied by subnormal galvanic reactivity. This is probably accounted for by the fact of central or peripheral exhaustion, and by the fact that in this condition the sweat-gland activity has reached its extreme physiological limit. The majority of the pathological mental conditions are characterized by subnormal electrical reactions. Prideaux has stated that dements have the least galvanic response, conversion hysterics slightly greater, the anxiety hysterics somewhat more, and normal individuals the most. Odegaard has observed that there is the least reactivity in the organic and schizophrenic psychoses, while 'neurotic' subjects and the 'constitutionally subnormal' show more, and normals the largest effects. According to Westburgh, various mental conditions give galvanic responses in the order of increasing magnitude as follows: paretics, catatonics, non-catatonic dementia præcox patients, manic-depressives, and normal persons. The impression is gained that the reduction in general galvanic reactivity in various mental conditions is more closely related to the severity of the attack than to qualitative differentiation. The traditional classifications are probably of but limited value for this sort of study, and when used should be handled with caution.

C. S. R.

PROGNOSIS AND TREATMENT

[63] **Relation of premature birth and under-weight condition at birth to mental deficiency.**—Aaron J. Rosanoff and Christine V. Inman-Kane. *Amer. Jour. Psychiat.*, 1934, **13**, 829.

A brief review of the more important contributions to the literature of the subject is given. The new material gathered by the authors reveals that among 122 institutional cases of mental deficiency there were 26, or 21·31 per cent., in which there was premature birth or under-weight condition at birth, or both. In a control group the corresponding percentage was only 3·89. The evidence seems conclusive to the effect that premature birth or under-weight condition at birth is an ætiological factor in mental deficiency. For the most part, if not entirely, the damage is caused by cerebral trauma occurring during birth. Premature and under-weight children are more liable to suffer such damage at birth than are children born at full term and of normal weight. Prematurity and under-weight condition at birth are more common in multiple than in single births; for that reason, and probably by the same mechanism, mental deficiency is produced more often in twins than in single births. Premature birth, or under-weight condition at birth, or both, do not cause mental deficiency per se. In the authors' group of cases no fewer than 19·29 per cent. of those in which the I.Q. had been ascertained showed an I.Q. of over 115.

C. S. R.

Reviews and Notices of Books

Histopathology of the Peripheral and Central Nervous Systems. By George B. Hassin, M.D., Professor of Neurology, University of Illinois, etc. London: Baillière, Tindall and Cox. 1934. Pp. 491. Price 30s.

DR. GEORGE HASSIN has long been known by repute for his numerous contributions to neuropathology, and it is fitting that he has now given us a textbook on a subject of which he is a foremost exponent. His views and theories on not a few controversial questions of neuropathology may not command universal acceptance, but at least they have the merit of originality. In this book he does not obtrude them unduly, being content rather to describe than to discuss; nevertheless he has succeeded in producing a work which has a character of its own, and which fills somewhat of a gap in the English literature of neuropathology, being larger than most primers dealing with the matter yet not unwieldy and, above all, not 'padded.'

On the whole, the presentation is worthy of the importance of the subject, though it is rather unequal; the chapters dealing with intracranial tumour, with inflammatory disease and syphilogenous disease are excellent; that on extrapyramidal disease is not so good—it lacks the critical touch, and contains expressions of opinion with which many will not agree and for which evidence is wanting. So far as we know no one has ever claimed that athetosis 'is a lesion of the corticospinal tract'; and it is inaccurate to aver that 'torsion spasm, athetosis, and spasmodic torticollis are practically one disease-process,' or that 'for a long time they were considered functional.' To state dogmatically that Schilder's disease is a 'morbid entity' rather overlooks the fact that its processes shade off by gradations to other disease-conditions. What the author writes of the pathology of pellagra stands in need of amplification and some revision. Little is said of congenital malformations and defects beyond descriptions of a few of the best known.

The book is finely illustrated and has useful bibliographies.

Die Parasagittalen Meningeome. By Dr. H. Olivecrona, Stockholm. With 145 illustrations. Leipzig: Georg Thieme. 1934. Pp. 144. Price M. 24.

THE lavish standard set by Cushing and others of his school in their series of neurosurgical monographs is maintained on the continent of Europe by this equally fine production by the Swedish authority Dr. Olivecrona. The work is based on some 34 examples of the particular tumour studied, which the author divides into three groups according as the site of growth has been the anterior, middle, or posterior third of the sagittal sinus. The clinical histories are recounted at some length, each being illustrated by drawings and X-ray

photographs, etc. A synthetic study of symptomatology and differential diagnosis follows, whilst some 20 pages are taken up with surgical technique. Of 33 cases submitted to operation, 15 per cent. ended fatally, and 10 per cent. in addition from recurrences. A complete or almost complete cure was effected in 50 per cent.; in 25 per cent. it was incomplete, restricting the earning or working capacity of the patients.

Abscess of the Brain: its Pathology, Diagnosis, and Treatment. By E. Miles Atkinson, M.B., B.S., F.R.C.S., Surgeon in charge of the Ear and Throat Department, Royal United Hospital, Bath, etc. London: Medical Publications Ltd. 1934. Pp. 289. Price 21s. net.

THE writer of this well-stocked monograph on brain abscess is to be congratulated on his performance. The book is based on wide personal experience of the condition with which it deals, and if the line of approach is self-evidently that of the otologist, rhinologist, or laryngologist rather than of the expert in nervous disease the fact matters little. Because of this approach the latter may think that some of the symptomatic descriptions (e.g., that of the speech disorder of left temporal lobe abscess) leave something to be desired, but at all events they are sufficiently detailed for practical purposes. Moreover, Mr. Atkinson has sought information where he can best find it, and has utilized the work of different neurologists in his accounts of cerebellar and other syndromes. The arrangement of the work is excellent, the illustrations (of which a number are in colours) are well reproduced, and statistical tables enhance the value of the book as a whole. Twenty-eight personal cases are chosen for publication at some length, and there is a good bibliography. Because of its intrinsic merits the book should have wide appreciation by both physicians and surgeons.

Psychopathology: a Survey of Modern Approaches. By J. Ernest Nicole, Senior Assistant Medical Officer, Lancashire County Mental Hospital, etc. Second edition, revised and enlarged. London: Baillière, Tindall and Cox. 1934. Pp. 283. Price 12s. 6d.

THE first edition of this attractive book was favourably reviewed in this JOURNAL (Vol. XII., p. 283). The second, somewhat enlarged and rewritten, impresses the reader even more than the original with the amount of information and critical examination of the tenets of different psychopathological schools that Dr. Nicole has been able to put in relatively small compass. He shows that the secrets of psychopathology are being approached to-day from analytical, neurological, psychological, physiological, and ethno-sociological standpoints, and affirms that not mere combination of such approaches, but their unification, may some day be possible. As we read the situation, that day is still somewhat remote. Instead of a multiple approach, not a few exponents of one or other line of attack scout the usefulness and deny the rationale of the remainder. Dr. Nicole himself does not perhaps clearly and

specifically indicate what his own view is—though doubtless he might regard this as of secondary importance; his purpose is to bring 'some sort of orderly disposition out of the chaos of modern theory.' In this respect he has succeeded admirably.

Textbook of Abnormal Psychology. By Roy M. Dorcus, Associate in Psychology, The Johns Hopkins University, and G. Wilson Shaffer, Professor of Psychology, University of Baltimore. London: George Allen and Unwin. 1934. Pp. 389. Price 16s.

THERE is no end to the writing of textbooks on abnormal psychology in these days, when the meaning of the word 'psychology' itself seems to have almost as many facets as there are authors who employ it. No little part of this work is devoted to pure neurology, for it contains chapters on sensory and motor disorders which belong to the field of the neurologist, and this is true also, in part at least, of that entitled 'Association Mechanism.' Workers in the allied fields of neurology and psychiatry have realized for years how intimate is their connexion, and are, or should be, the last to enforce an artificial separation. But while each should know much of the other's sphere, we cannot accept the views expressed by the authors of this textbook, who baldly state that 'in its narrower meaning the function of the brain and neural pathways is outside of' the sphere of neurology (*sic*), nor will neurologists concede their 'definition' of neurology, viz., that it 'is a study of the structure and growth of the nervous system, including the brain, spinal cord, and receptors of the special senses.' Neurology has a vastly wider connotation, and properly so. The authors allege that in the case of 'glove anæsthesia' 'the neurologist can definitely say that it is not organic' (they are unaware that this type of sensory loss is found also in some 'organic' forms of nervous disease); 'the neurologist cannot say, however, what factors are involved in producing the condition, nor prescribe the treatment to be administered, since no medicament has been discovered for treating such cases.' It is to be deplored that so singular a misrepresentation of neurology should find a place in a work of this kind.

From the standpoint of psychopathology, however, the book can be read with profit; its exposition of 'functional psychoses,' psychotherapy, and mental deficiency is satisfying and well set forth. Under the heading of Psychoneuroses we find a description of epilepsy, for no other reason, seemingly, than that the epilepsies are imagined by the writers to have a 'frequent similarity to hysterical states.' Here the authors are guilty of an unfortunate classification. Like some other psychopathologists, they lay unwarranted stress on the occurrence in cases of 'genuine epilepsy' (a misleading term) of 'certain definite personality traits' present before convulsive seizures appear. Their discussion of sleep and its basis is interesting, yet hardly does justice to some present-day physiological research; they do not make convincingly clear the relation of sleep to 'abnormal psychology.'

Mental Defect. By Lionel S. Penrose, Research Medical Officer, Royal Eastern Counties Institution, Colchester. London: Sidgwick and Jackson. 1934. Pp. 183. Price 8s. 6d. net.

THE latest addition to the textbooks of social biology edited by Professor Lancelot Hogben is concerned with mental defect in its varying aspects. Dr. Penrose is well qualified by experience and amount of material at disposal to deal with these, which he does in an engaging and informative manner. The chapters on systematic investigation cover all the present-day methods of study of the mental defective, and if they contain nothing novel at least they illustrate the importance of attention to detail. A good deal of trouble has been expended on exposition of analytical methods, e.g., on Mendelian ratios and gene frequencies. The author criticizes present-day classification, regarding with some favour that recently proposed by Lewis, which divides mental defectives into 'subcultural' and 'pathological' groups. Ætiology, pathological types, and treatment are all dealt with at some length. It is regrettable that the volume has no index to its contents, only one of names and authors being supplied. There is, however, a glossary, doubtless provided for those 'lay persons' mentioned in the preface who desire knowledge on 'some of the scientific problems associated with the study of mental deficiency.' If such persons do not know the meaning of 'morbid,' 'cutaneous,' 'occiput,' 'reflex,' and other terms obligingly defined by the author, it is absurd to imagine they are capable of understanding the 'scientific problems' to which he makes reference. Further, in some of the definitions of this glossary words are used that stand in need of definition themselves, e.g., thyroid, cortex, pituitary, pelvis, etc.—that is, if we judge by the standard of ignorance that is presumed. Again, technical terms used in the text, such as midbrain, neuroepithelioma, meningocœle, umbilical hernia, and numerous others find no place in the glossary. Why the author should cater for sciolism in a professional work of this kind we are at a loss to understand.

To Be or Not to Be: A Study of Suicide. By Louis I. Dublin, Ph.D., Statistician and Third Vice-President of the Metropolitan Life Insurance Company. New York: Harrison Smith and Robert Haas. 1934. Pp. 443. Price $3.50.

WE can cordially recommend this treatise on the perennial problem of suicide to the notice of our readers. It is packed full of information drawn from widely differing sources; its discussions are dispassionate, its attitudes scientific. Complex factors of race, age, sex, of intrinsic personality and extrinsic circumstances, are fully analysed, and many tables add to the value of the critical dissection of the matter. There is much, too, of historical and religious significance, while the style of writing is pleasant, with literary quotations that add to the general interest of the work. The legal aspects of the question are examined, as well as those bearing on life assurance; Suicide and Mental Disease is the title of one chapter, and another is devoted to the

preventive side of the problem. The book ends on a note of encouragement, in a chapter entitled ' Towards Sound Mental Health.' Numerous appendices and a good bibliography complete a most useful and comprehensive study.

Diffuse Sclerosis (encephalitis periaxialis diffusa). By L. Bouman, M.D., Professor of Psychiatry and Neurology, Utrecht University. With 64 illustrations. Bristol: John Wright and Sons. 1934. Pp. 160. Price 15s. net.

PROFESSOR BOUMAN'S monograph on diffuse sclerosis is sure of being appreciated by students of nervous disease. It covers the field excellently in accordance with tradition by ranging from a historical introduction, through clinical and pathological descriptions, to differential diagnosis. Unfortunately, in the present state of knowledge little can be said of prognosis and treatment. Tabulated notes of 100 cases are provided in summary at the close of the book.

The author suspends judgement on the debatable point of whether the lesions are inflammatory or degenerative, though he allows that some recorded examples definitely seem to belong to the former category and others to the latter. He also provides an interesting excursus on the subject of pathogenesis and on the relation of lipoids to the dystrophic glial process. The illustrations are well reproduced and the bibliography is fairly complete.

Une Nouvelle Syphilis Nerveuse. By Paul Ravaut, Member of the Academy of Medicine, Paris. Paris: Masson et Cie. 1934. Pp. 196. Price 45 fr.

THE ' new ' nervous syphilis of M. Ravaut has reference to the years since the beginning of the century, when cytodiagnosis of the spinal fluid opened a new world to the student of neurosyphilis. These years have witnessed many additions to knowledge of the subject, but M. Ravaut has done well in concentrating here on clinically latent or non-evident forms, disclosed only by the findings in the fluid. This aspect of neurosyphilis has engaged his attention for a very long time now, and his treatise embodies the results of his investigations. The term ' biological neurosyphilis ' is suggested for ' preclinical ' neurosyphilis, when only the fluid throws light on the processes that are going on. A chapter of practical importance deals with the prognosis of latent cases; a large number of statistics are cited which, combined, show that among 825 syphilitics having a negative fluid in the course of the primary disease only 20 later showed clinical or laboratory signs of nervous syphilis, or 2·4 per cent. The interpretation of positive findings in the course of latent syphilis is difficult; its prognostic meaning will depend on the age of the syphilis, previous treatments, coexisting blood reactions, etc.; an endeavour should be made to ascertain whether the finding represents the active evolution of a process or of one that is becoming extinct. This question has to be decided by the qualities of the reaction itself.